A Review of the Scientific Literature As It Pertains to Gulf War Illnesses

VOLUME 6

OIL WELL FIRES

Dalia M. Spektor

National Defense Research Institute

RAND

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Dalia M. Spektor

Prepared for the Office of the Secretary of Defense

National Defense Research Institute

RAND

PREFACE

This literature review, one of eight commissioned by the Special Assistant to the Deputy Secretary of Defense for Gulf War Illnesses, summarizes the existing scientific literature on the health effects of the oil fires that may have affected military personnel who served in Operations Desert Shield and Desert Storm. The eight RAND reviews are intended to complement efforts by the Defense Department and other federal agencies in their attempt to understand the full range of health implications of service in that conflict.

Many veterans have reported an array of physical and mental health complaints since the war. Whether veterans are experiencing either higher-than-expected rates of identifiable illnesses with known etiologies or other illnesses from unidentified origins is not yet clear.

The other seven RAND literature reviews deal with chemical and biological warfare agents, depleted uranium, pesticides, pyridostigmine bromide, immunizations, infectious diseases, and stress. These represent plausible causes of some of the illnesses Gulf War veterans have reported.

The reviews are intended principally to summarize the scientific literature on the known health effects of given exposures to these risk factors. Where available evidence permits, the reviews also summarize what is known about the range of actual exposures in the Gulf and assess the plausibility of the risk factor at hand as a cause of illnesses. Statements related to the Gulf War experience should be regarded as suggestive rather than definitive, for more research on health effects and exposures remains to be completed before definitive statements can be made. Recommendations for additional research where appropriate are included.

These reviews are limited to literature published or accepted for publication in peer-reviewed journals, books, government publications, and conference proceedings. Unpublished information was occasionally used, but only to develop hypotheses.

This work is sponsored by the Office of the Special Assistant and was carried out jointly by RAND Health's Center for Military Health Policy Research and the

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SUMMARY

BACKGROUND

When the Iraqi army withdrew from Kuwait, it left the Kuwait oil fields in flames. Over half of the more than 1000 oil wells were burning, a most spectacular and unforgettable sight. The sheer magnitude of the fires caused concern about potential global effects, including alteration of worldwide weather patterns and a drastic increase in acid rain. Fortunately, these concerns proved excessive; the fires were extinguished much sooner than originally anticipated or thought possible. Although the fires could be detected by sensitive instruments several thousand miles from the Persian Gulf, the global catastrophe many predicted did not occur. The fire characteristics were such that the plumes were not energetic enough to pierce into the upper atmosphere and cause long-term climatic changes.

Because of the proximity of U.S. troops, concerns about the effects of the fires on them were also high. Burning crude oil produces a wide range of pollutants such as carbon dioxide, carbon monoxide, sulfur dioxide, nitrogen oxides, volatile organic compounds (VOCs) (e.g., benzene), polycyclic aromatic hydrocarbons (PAHs) (compounds such as anthracene that form during the incomplete combustion of organic substances), hydrogen sulfide, acidic gasses (e.g., sulfuric acid), and soot. Several of these pollutants have been linked with short- and long-term illnesses, including upper-respiratory ailments and a variety of cancers.

The geographic and climatic characteristics of the Gulf region exacerbated the hazard posed by the pollutants. Most of the area is desert, and the sandy soils of the region are a boundless source of particulate matter, some of which is very fine. The region is subject to high winds, especially in the spring, that stir up large amounts of fine sand dust endemic to the region. Not only does the particulate matter pose a health risk in its own right, but it could also serve as a carrier for the airborne pollutants generated as a by-product of the oil fires.

Particles smaller than 2.5 microns[1] pose a special threat because they can penetrate deeply into the lungs, and recent studies have found strong associations between fine particles and premature death and morbidity from cardiopulmonary diseases when compared with larger particles.

MONITORING EFFORTS

No systematic environmental monitoring took place in the region from the initial deployment in August 1990 until May 1991. To determine the extent of the health hazard of the oil fires to U.S. forces and others, several monitoring teams were sent. The team from the U.S. Army Environmental Health Agency (USAEHA), presently known as the U.S. Army Center for Health Promotion and Preventive Medicine (CHPPM), carried out the most extensive series of measurements, monitoring both air and soil pollution. From May until December 1991, the USAEHA team collected about 4000 samples. When the team began its sampling, only about 8 percent of the fires had been put out, and the team continued its work until after all fires were extinguished. The team sampled soil and air at eight sites in Kuwait and Saudi Arabia, primarily in locations with large concentrations of U.S. personnel.

In addition to the USAEHA team, several other groups carried out environmental sampling. These efforts were not as comprehensive as those of the USAEHA team. For example, one project measured particulate matter in Bahrain from late July to early August 1991. The U.S. Environmental Protection Agency (EPA) sent a team of experts to monitor oil-fire emissions during March 13–20 and March 24–27 when most fires were still burning. Another tested for polycyclic aromatic hydrocarbon compounds and trace metals at ground level and inside smoke plumes. The measurements taken by these other groups confirmed the data taken by the USAEHA team.

HEALTH EFFECTS STUDIES

Studies were conducted during the Gulf War to assess possible health effects due to emissions from the Kuwait oil well fires. VOC levels in the blood of U.S. personnel in Kuwait City were measured in May 1991 and found to be equal to or lower than a control group residing in the U.S. Similar measurements on firefighters working in the oil fields in October 1991 yielded levels about 10 times higher than the control group. No difference in genotoxicity was found between soot brought from Kuwait and air particulates isolated from Washington, DC.

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[1]A micrometer or micron is 1/1,000,000 of a meter.

Specimens from the lungs, liver, kidneys, and blood and urine samples of Kuwaiti feral cats were examined. Except for minimal changes in the larynx, no lesions were found that could be attributed to breathing smoke from the oil well fires or associated with hydrocarbon inhalation.

Several self-reported health surveys were conducted on Gulf War veterans. Symptoms questionnaires were completed before, during, and after deployment by the 11th Armored Cavalry Regiment based in Fulda, Germany. The symptoms that appeared during their stay in Kuwait but were not experienced before were headache (55 percent), lightheadedness (48 percent), fatigue or weakness (45 percent), skin rashes (41 percent), and diarrhea (42 percent). About 35 percent of those who did not usually cough or have phlegm first thing in the morning before deployment reported the symptom after arriving in Kuwait. Symptoms were associated with reported proximity to the oil well fires, and their incidence generally decreased after soldiers left Kuwait.

The Navy's self-administered symptoms questionnaires were completed by three groups of U.S. Marines. The group that had the longest exposure and was located closest to the oil fires reported a higher rate of gastrointestinal episodes, respiratory symptoms, and burning and red eyes. The prevalence of wheezing, coughing, runny nose, and sore throat for each group decreased with increased distance from the fires. Smokers reported more complaints than nonsmokers.

The Iowa Persian Gulf Study reported a significantly higher prevalence of self-reported symptoms among military personnel deployed during the Gulf War compared with those on active duty at the same time but not deployed to the Gulf. Larger differences between Gulf War and non–Gulf War veterans were observed for National Guard and Reserve components. Results from this study on the prevalence of symptoms of airway disease indicate a statistically significant rate difference of 2.3 percent for asthma and bronchitis between Gulf War and non–Gulf War military personnel.

RESULTS

The concentrations of VOCs, polycyclic aromatic compounds, metals, and criteria pollutants were much lower than initially presumed, considering the magnitude of the fires. The mean-concentration measurements of these pollutants are consistent across studies. The maximum concentrations measured in the Persian Gulf region are virtually the same levels found in suburban locations in the United States, lower than those found in large urban centers in the United States, and much lower than the U.S.-recommended occupational levels.

The data show that the concentration of the pollutants present in the environment as a consequence of the oil well fires fell below the exposure limits for

hazardous substances in the workplace recommended by the National Institute of Occupational Safety and Health, Occupational Safety and Health Administration, or American Conference of Governmental Industrial Hygienists. For example, the mean benzene concentration measured in Ahmadi, Kuwait, was 7.8 $\mu g/m^3$; this compares to the ACGIH occupational standard of 1600 $\mu g/m^3$ for an 8-hour-day, 40-hour-week workshift. One who smokes two packs of cigarettes a day inhales about 1200 μg of benzene, compared to 150 μg a day of ambient benzene inhaled by nonsmokers in Ahmadi. In most cases, the ambient air concentrations of pollutants in areas where military and civilian personnel were located were found to be orders of magnitude lower than those in occupational settings in the United States and similar to ambient levels.

The levels of pollutants measured in the Gulf were much lower than those that are known to cause short- or long-term health effects. The intensity of the fires made combustion relatively efficient, and the presence of coarse particles in the clouds assisted in removing other pollutants from the smoke plumes.

The health surveys conducted during the oil fires indicate increased symptoms and an association between prevalence of complaints and proximity to the fires. No data confirm, however, that those symptoms were indicators of disease.

Particulates are another story. Measurements at all monitoring sites show that particulate concentrations were much higher than ambient levels in the U.S. The high density of atmospheric particles did not result from the oil fires; rather, it is characteristic of the region itself. Comparison of measurements taken in 1991 and in 1994, when the fires had long been extinguished, show similar average values. Results of the relatively few measurements of particle size indicate that there was a significant amount of particles in the size range that can have an effect on the respiratory system of sensitive subgroups, e.g., smokers, or those who have a history of asthma or bronchitis. Thus, U.S. troops could have been exposed for several months to concentrations of fine particulate matter (smaller than 2.5 μm) that exceed U.S. air-quality standards. Although some personal communications indicate increased respiratory complaints among the indigenous population during the oil fires, no epidemiological studies or evidence of health effects were found in the peer-reviewed literature.

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ACRONYMS

ACGIH	American Conference of Governmental Industrial Hygienists
BS	British smoke
CBD	Chronic beryllium disease
CCEP	Comprehensive Clinical Evaluation Program
CHPPM	US Army Center for Health Promotion and Preventive Medicine
COPD	Chronic obstructive pulmonary disease
EPA	Environmental Protection Agency
IARC	International Agency for Research on Cancer
MRL	Minimum risk levels
NAAQS	National Ambient Air Quality Standard
NASA	National Aeronautics and Space Administration
NIOSH	National Institute of Occupational Safety and Health
NOAA	National Oceanic and Atmospheric Administration
OSHA	Occupational Safety and Health Administration
PAH	Polycyclic aromatic hydrocarbons
PBL	Planetary boundary layer
PEL	Permissible exposure limit
PTSD	Post traumatic stress disorder
RBC	Red blood count
RR	Relative risk
TLV	Threshold limit value
TSP	Total suspended particulates
TWA	Time-weighted average
USAEHA	U.S. Army Environmental Hygiene Agency
VOC	Volatile organic compounds

Chapter One

INTRODUCTION

PURPOSE OF THE REPORT

The purpose of this work is to conduct a comprehensive scientific literature review on the possible health effects due to exposures to the Kuwait oil well fires and to produce a stand-alone, short scientific report on the findings that would pass peer-review scrutiny.

Concerns about the possible health effects experienced by the troops deployed in the Gulf due to exposure to emissions from the oil fires triggered an extensive effort to determine the extent of those environmental exposures. That effort yielded quantitative data on what pollutants were present in the Gulf and at what concentrations.

The target audience for the present report is the scientific community familiar with the issues, methodologies, and processes that are the basic tools of environmental health science. The report contains a summary geared for a policy-maker.

This work involved comprehensive library searches that yielded about 2500 titles. Of these, about 500 abstracts were examined and then reduced to approximately 250 peer-reviewed papers that were analyzed. About 180 of these are cited in this report. Also, about 35 pertinent reports from U.S. and international agencies and institutions were reviewed.

ORGANIZATION OF THE REPORT

A brief introductory chapter provides a concise description of the geographical characteristics of the Gulf region, the U.S. troop presence there during Operations Desert Shield and Desert Storm, and the characteristics of the Kuwait oil fires.

The introduction is followed by a chapter reviewing the U.S. air quality standards and recommended limits for chemical hazards, and the environmental measurements available for the Gulf region before, during and after the Kuwait oil well fires. This chapter establishes the list of pollutants present and their concentration levels, shows the consistency of these results across studies, and compares the prevalence of the pollutants to U.S. ambient measurements and occupational standards. The first two chapters are a necessary background for the stand-alone report described above and in no way presume to be either a critical or comprehensive analysis and evaluation of all environmental data.

The third chapter describes the possible health effects that could be associated with pollutants present in the Gulf region. Because of the volume of references on many pollutants, the presentation is limited to possible health effects for exposures closest to those measured in the Gulf. The last section summarizes the results of several health studies related to Gulf War veterans. It also includes an overview of the veterans' symptoms information to complete the stand-alone report and in no way presumes to be either a critical or comprehensive review.

The final chapter presents the findings and conclusions of the present literature review based on the exposures measured in the Gulf region. It discusses what health effects are plausible at the exposure levels measured in the Gulf, how those compare with the list of veterans' symptoms, and what areas need further research to elucidate the relationship between exposure and effect.

BACKGROUND

Iraq invaded Kuwait on August 2, 1990. In support of United Nations Resolution 660, the United States responded by sending troops to the Persian Gulf in Operation Desert Shield. On January 16, 1991, Operation Desert Storm began with an air war against Iraq that was followed, 39 days later, by a four-day ground war. By the time hostilities concluded, 697,000 American troops had served in the Persian Gulf. Of that number, 17 percent were from the Reserve Components, and 7 percent were women. Although nearly 700,000 troops passed through the theater, the peak number there at any one time was about 560,000.

As the Iraqis withdrew from Kuwait, they set fire to over half of Kuwait's 1000 oil wells and damaged most of the rest. Industry experts estimated at the time that these fires were burning 5–6 million barrels of crude oil per day and 70–100 million m^3 per day of natural gas. The greater Al Burqan oil field, with approximately 700 wells (of which 365 were ignited), was the most important field in terms of oil production, number of wells on fire, and amount of smoke generated. Oil in the field continuously rose to the surface due to high subsurface pressure. This field probably had the greatest effect on exposed humans since it

was the closest to Kuwait City and other coastal communities. The first fires were extinguished in early April 1991 and the last fire was extinguished on November 6, 1991. Figure 1.1 shows the relationship between U.S.-troop presence in the Gulf and the oil fires (USAEHA, 1994). It also shows when most of the air-quality monitoring occurred.

The Arabian Peninsula

The region's geography and climate are important to any consideration of the effects of the oil well fires. Saudi Arabia occupies most of the Arabian peninsula. It consists of about 2,150,000 km^2 of plains with scant vegetation (except near oases), and has a population of about 16 million. Kuwait lies between Saudi Arabia, Iraq, and the Persian Gulf. It has about 18,000 km^2 of sandy desert and coastal beaches, and had an estimated population of about 800,000 natives and 1,300,000 immigrants as of 1990 (Husain, 1995).

The weather in Kuwait and eastern Saudi Arabia is typical of the Sahara region. It is characterized by long, hot, and dry summers; it also has short, warm, and sometimes rainy winters, with 4 inches of rain in Kuwait and 1 inch in coastal Saudi Arabia. The region is subject to violent storms. It also is prey to fierce winds (called Shammal winds) that blow sand and dust that can reduce daytime visibility to a few meters. Average winds in Kuwait are from the northwest at approximately 14 mph. The highest recently recorded temperature was 51°C (124°F) in July 1978; the lowest was –6°C (21°F) in January 1964. Temperatures

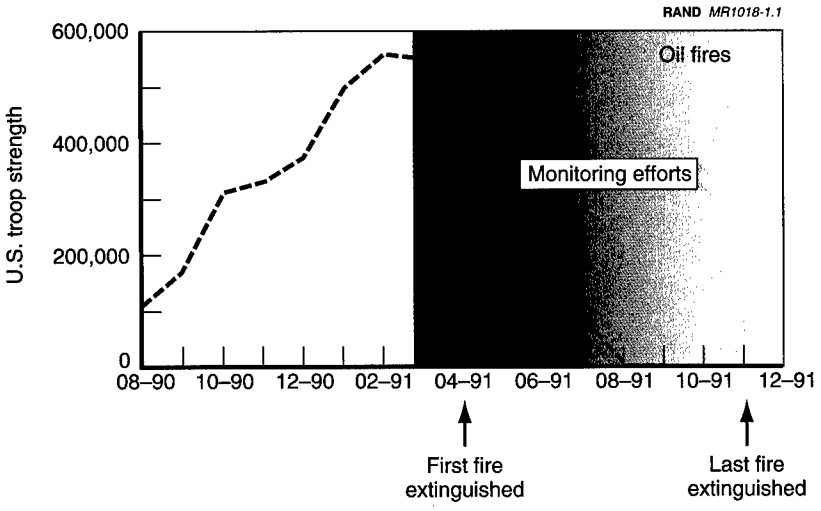

Figure 1.1—Kuwait Oil Fires and U.S. Troop Presence

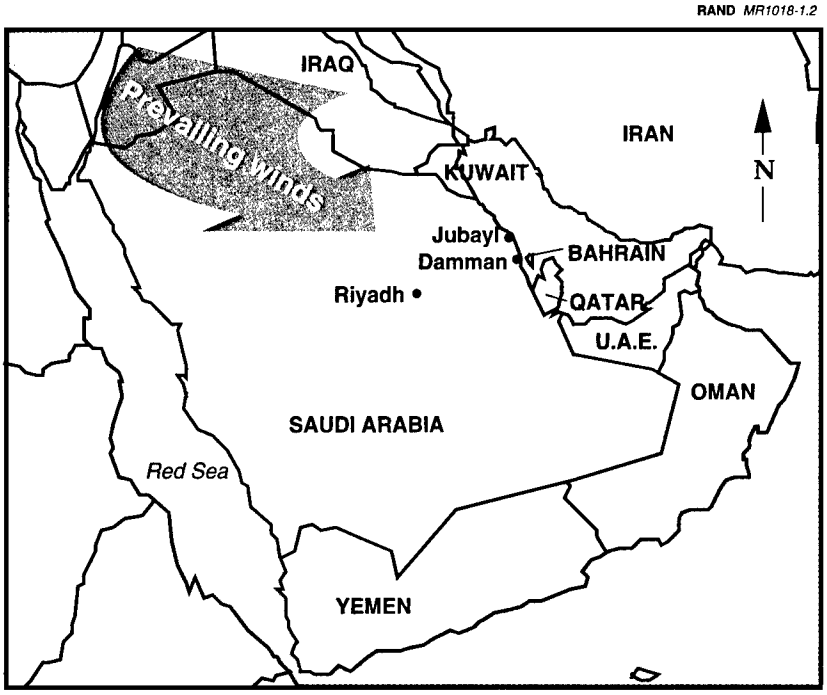

Figure 1.2—Gulf War Region

also vary widely by season, ranging from an average of 45°C (113°F) in summer to an average of 8°C (46°F) in winter (Nasralla, 1983).

Kuwait Oil Well Fire Characteristics

Typically, burning crude oil produces smoke containing a large number of particulate and gaseous species such as carbon dioxide (CO_2), carbon monoxide (CO), sulfur dioxide (SO_2), nitrogen oxides (NO_x), volatile organic compounds (VOCs), polycyclic aromatic hydrocarbons (PAHs), hydrogen sulfide (H_2S), acidic aerosols, and soot.[1]

However, the composition of crude oil differs by strata and region. Calculations based on airborne measurements of the smoke from the Kuwait oil fires in May and June 1991 (Ferek, 1992) indicate that the combined oil and gas emissions were equivalent to the burning of approximately 4.6 million barrels of oil per day, somewhat less than estimated by industry experts. The combustion was

[1]Soot: A conglomeration of particles impregnated with tar, formed by the incomplete combustion of carbonaceous material.

relatively efficient; about 96 percent of the fuel carbon burned was emitted as CO_2. Particulate smoke emissions averaged 2 percent of the fuel burned, of which approximately 20 percent was soot. About two-thirds of the mass of the smoke was accounted for by salt, soot, and sulfate. The salt most likely originated from oil-field brines that were ejected from the wells along with oil. These oil brines are formed from salt deposits in certain layers of the wells, which penetrate the Jurassic strata typically 1.5–3.0 km below the surface (Cahalan, 1992). Crude oil may contain nickel, vanadium, arsenic, and other metals in small or trace quantities, many of which are removed during refining. General characteristics of Kuwait crude oil appear in Table 1.1.

Although the composite smoke plumes in satellite photographs appeared black, individual fires emitted plumes with a variety of colors and densities. About 25 percent of the plumes were white or light gray and others black or dark gray. The densest black plumes were from large ignited pools of oil on the ground. The white plumes contained higher concentrations of NaCl and CaCl crystals (many in the fine fraction (<2.5 μm) range) and of SO_2 than the black plumes (Stevens, 1993).

Initial calculations of worst-case effects of the burning oil wells assumed about a 10 percent soot content (Bakan, 1991). This level of soot could alter not only the region's climate but also perhaps that of the entire globe (Bakan, 1991). Actual measurements showed a much lower concentration. The soot concentration was only 0.45 percent of the fuel burned, or 0.53 percent of the total carbon emitted (Ferek, 1992). This measurement is over an order of magnitude lower than the 10-percent estimate (Bakan, 1991).

Fear of large emissions of SO_2 caused a local health-hazard concern, as well as concern that long-range transport of the smoke might produce acid rain glob-

Table 1.1

Characteristics of Kuwait Crude Oil

Component	Amount
Sulfur	2.44 %w
Nitrogen	0.14 %w
Nickel	7.7 ppm
Vanadium	28 ppm
Low-boiling point naphtha $\leq 205^{o}C$	22.7 %w
High-boiling point naphtha $\geq 205^{o}C$	77.3 %w
Aromatics	23.3 %w
Paraffins	20.9 %w
Insolubles	3.5 %w

SOURCE: Ferek, 1992.

NOTES: %w = percent by weight; ppm = parts per million.

ally. Partly because of the early and successful extinction of the oil fires, these concerns did not materialize. Measurements of the removal rates of SO_2 and NO_x after emission show that, on average, they were 6 percent per hour for SO_2 and 22 percent per hour for NO_x. A likely explanation for the rapid removal of SO_2 and NO_x is through reactions with coarse-mode sand dust. The large concentrations of soil dust probably scavenged SO_2 and NO_x from the plumes (Hobbs and Radke, 1992).

Natural winds tended to move smoke plumes toward the southeast, but occasionally a plume moved toward the northeast, merging with a southeast-bound plume and creating a composite or "superplume" that continued southeast.

Through the summer, there were local inversion episodes, and the plumes could not rise. Such inversions may have reinforced themselves by keeping the smoke optically thicker than it would otherwise be, thus leading to more solar absorption in the atmosphere and cooling below. This effect helped to maintain the confinement (Cahalan, 1992). The smoke plumes were never observed to rise above 6 km, even after traveling 1600 km in 48 hours. In general, the plumes were depleted and dispersed for several thousand miles downwind over a period of several weeks, but always well below the base of the stratosphere (approximately 13 km). Such altitudes are low enough to prevent global distributions of the smoke-plume contaminants. Therefore, the effects of the contaminants were limited to the region, although the diluted plumes were detected worldwide.

Sometimes local atmospheric inversion patterns returned high concentrations of pollutants to ground level (Hobbs and Radke, 1992). Smoke particles in the plume coagulated as they were transported downwind and became hydrophilic as a result of being coated with sulfate. This substantially increased their removal efficiency by clouds and rain. Within a few kilometers of the fires, the circulation generated by the heat of combustion carried the pollutants aloft, with relatively clean air flowing into the fire region from all sides. Heavy particles and unburned oil droplets, large enough to be visible to the naked eye, fell through this clean air and caused a visible "plume" that was largely not associated with the combustion products.

The smoke plumes were 15 to 150 km wide from the source up to distances of 1000 km from the fires. The plumes absorbed sunlight, making the ground surface colder and darker. Daytime temperatures below the plumes were reportedly 10°C or more below normal under the optically thickest part of the plume, within 100 km of the source (Cahalan, 1992). Despite the darkness, the air at ground level retained relatively low pollution levels, except during the in-

version episodes. Satellite images indicated that frequent intense dust storms mixed with the smoke in the same region either at the same level or in stratified layers (Limaye, 1992).

Chapter Two

ENVIRONMENTAL MEASUREMENTS

This chapter reviews U.S. ambient-air-quality standards and recommended limits for chemical hazards, as well as environmental measurements available for the Gulf region before, during, and after the Kuwait oil well fires. This chapter describes the concentration levels of pollutants measured during the oil fires, shows the consistency of these measurements across studies, and compares their magnitude to U.S. ambient air and occupational standards. This chapter provides the necessary background for this to be the stand-alone report described in the introduction. It is an overview and in no way presumes to be either a critical or comprehensive analysis and evaluation of all the environmental data. For a more detailed discussion of the environmental data presented in the following tables, refer to the source references. The target audience for the present chapter, as mentioned above, is the scientific community familiar with the issues, methodologies, and processes that are the basic tools of environmental health science.

The magnitude of the fires and the extensive publicity about the amount of smoke sparked concern around the world regarding effects on global processes such as ozone depletion, acid rain, global warming, and other atmospheric phenomena. For example, some worried that the smoke could disrupt the monsoon season in southern Asia and trigger a drought affecting crops in India and Pakistan (Horgan, 1991a).

Although no global effects were noted, the passage of the smoke was detected worldwide where very sensitive equipment was located. Beginning in February 1991, the National Oceanic and Atmospheric Administration (NOAA) recorded numerous "soot spikes" in air-sampling data at its observatory located 4000m above sea level in Hawaii. Wind-pattern records indicate that the sooty air had left the Persian Gulf 7–10 days earlier. The March levels at Mauna Loa were five times higher than during March in the previous three years; these levels were still low enough to pose negligible health risks. No global disaster occurred, as some predicted immediately after the fires began (Horgan, 1991a, 1991b).

Firefighters started to cap the flaming oil wells in April, expecting that they would need up to two years to extinguish all fires. In the meantime, a coordinated international research effort began in the spring of 1991 to assess the impact of the fires on health and the environment.

U.S. STANDARDS AND RECOMMENDATIONS

The 1970 Clean Air Act mandates the U.S. Environmental Protection Agency (EPA) to promulgate standards and risk assessments for the chemicals that, above certain exposure levels, may generate health risks for the general population. The standards focus on the pollutants' toxicity, including carcinogenicity, and on estimated release volumes. A small number of pollutants, called Criteria Pollutants, are ubiquitous in the United States, i.e., most people are exposed to them daily, and extensive research into their health effects has been accumulated. The Clean Air Act requires the EPA to: (1) review public health standards for six major air pollutants (the Criteria Pollutants) every five years (2) update the standards, if necessary, to "protect public health with an adequate margin of safety," based on the latest, best-available science (3) consider only the public health, and not the cost of compliance, when setting the air-quality standards, and save cost considerations for the implementation phase. The Criteria Pollutants are ozone (O_3); particulate matter of less than 10 micrometers in diameter (PM_{10}) and of less than 2.5 micrometers in diameter ($PM_{2.5}$); carbon monoxide (CO); nitrogen dioxide (NO_2); lead (Pb); and sulfur dioxide (SO_2). The data on these pollutants are integrated by the EPA to set a National Ambient Air Quality Standard (NAAQS) for each criteria pollutant. These standards are listed in Table 2.1.

Table 2.1

Federal Ambient Air Quality Standards

Pollutant	Averaging Time	Concentration	
Ozone (O_3)	8 hour	0.080 ppm	(160 $\mu g/m^3$)
Particulate Matter			
PM_{10}	Annual mean	50 $\mu g/m^3$	
	24 hour	150 $\mu g/m^3$	
$PM_{2.5}$	Annual mean	15 $\mu g/m^3$	
	24 hour	65 $\mu g/m^3$	
Carbon Monoxide (CO)	8 hour	9 ppm	(10 mg/m^3)
	1 hour	35 ppm	(40 mg/m^3)
Nitrogen Dioxide (NO_2)	Annual mean	0.053 ppm	(100 $\mu g/m^3$)
Lead (Pb)	Calendar quarter	1.5 $\mu g/m^3$	
Sulfur Dioxide (SO_2)	Annual mean	0.030 ppm	(80 $\mu g/m^3$)
	24 hour	0.14 ppm	(365 $\mu g/m^3$)

SOURCE: EPA NAAQS, 1997.

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) requires that the Agency for Toxic Substances and Disease Registry (ATSDR) develop jointly with the EPA a list of the most common hazardous substances and prepare a toxicological profile for each substance. The ATSDR Minimal Risk Levels (MRL) were developed as the response to this mandate. An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over the specified exposure duration. MRLs are generally based on the most sensitive substance-induced end point considered to be of relevance to humans (ATSDR, 1997). Table 2.2 lists the available MRLs for the pollutants of concern.

The National Institute for Occupational Safety and Health (NIOSH) was established by the Occupational Safety and Health Act of 1970 as part of the Centers for Disease Control (CDC). One of NIOSH's mandates is to recommend criteria for preventing disease and hazardous conditions in the workplace. NIOSH develops and periodically revises recommended exposure limits—i.e., time-weighted average concentrations (TWAs)—for hazardous substances by evaluating all available medical, biological, and other relevant information. These recommendations are transmitted to the Occupational Safety and Health Administration (OSHA) and the Mine Safety and Health Administration for use in promulgating legal standards. NIOSH's TWA standards are the maximum recommended exposures in the workplace for up to a 10-hour workday during a 40-hour workweek. OSHA's TWA concentrations must not be exceeded during any 8-hour workshift of a 40-hour workweek.

Table 2.2

ATSDR Minimal Risk Levels for Hazardous Substances

Substance	Duration	Concentration	Endpoint
Benzene	Acute	0.5 ppm (1600 $\mu g/m^3$)	Immunological
Toluene	Acute	3 ppm (11,500 $\mu g/m^3$)	Neurological
	Chronic	1 ppm (3800 $\mu g/m^3$)	Neurological
Xylene	Acute	1 ppm (4400 $\mu g/m^3$)	Neurological
	Intermediate	0.7 ppm (3100 $\mu g/m^3$)	Developmental
	Chronic	0.1 ppm (440 $\mu g/m^3$)	Neurological
Cadmium	Chronic	0.2 $\mu g/m^3$	Renal
Chromium (III)	Intermediate	0.02 $\mu g/m^3$	Respiratory
	Chronic	0.02 $\mu g/m^3$	Respiratory
Mercury	Acute	0.02 $\mu g/m^3$	Developmental
	Chronic	0.014 $\mu g/m^3$	Neurological
Nickel	Intermediate	0.1 $\mu g/m^3$	Respiratory
Vanadium	Acute	0.2 $\mu g/m^3$	Respiratory

SOURCE: ATSDR, 1997.

The American Conference of Governmental Industrial Hygienists (ACGIH), a professional society—not a government agency—devoted to the administrative and technical aspects of occupational and environmental health, develops Threshold Limit Values (TLVs) as recommendations or guidelines for use in the practice of industrial hygiene in the workplace. TLVs are TWA concentrations that should not be exceeded during any 8-hour workshift of a 40-hour workweek. All these concentration limits are set at levels believed to be without adverse effects for nearly all workers during repeated daily exposure. Table 2.3 lists NIOSH, OSHA, and ACGIH recommended occupational limits for the noted pollutants.

Since the EPA's standards are set to protect the general population—including those most vulnerable, such as small children, the elderly, and people with

Table 2.3

NIOSH, OSHA, and ACGIH Occupational Limits for the Pollutants of Interest ($\mu g/m^3$)

Pollutant	Symbol	NIOSH[b]	OSHA[b]	ACGIH[b]
Ozone	O_3	200	200	100
Sulfur dioxide	SO_2	5000	13,000	5200
Nitrogen dioxide	NO_2	1800[c]	9000[c]	5600
Nitric oxide	NO	5000	5000	31,000
Acenaphthene[a]		100	200	200
Benzo(a) anthracene[a]		100	200	200
Biphenyl[a]		100	200	200
Chrysene[a]		100	200	200
Fluoranthene[a]		100	200	200
Phenanthrene[a]		100	200	200
Pyrene[a]		100	200	200
Particulate	PM_{10}	NA	NA	3000[d]
Cadmium	Cd	NA	5	10
Chromium	Cr	500	500	500
Nickel	Ni	NA	1000	120
Lead	Pb	100	50	50
Vanadium	V	50	50	50
Zinc	Zn	1000	1000	500
Benzene		320	3200	1600
Toluene		750,000	375,000	188,000
Ethyl benzene		435,000	435,000	435,000
m,p-Xylene		435,000	435,000	435,000
o-Xylene		435,000	435,000	435,000

SOURCES: NIOSH, 1997; ACGIH, 1997.

NOTE: NA = Not applicable.

[a]Components of coal tar pitch volatiles. [b]Concentration limits are for their respective TWAs except for NO_2. [c]Short-term exposure limit, a 15-minute TWA. [d]PM_{10} not otherwise regulated.

respiratory and other diseases—from increased health risks due to exposures to ambient pollutants, they are much lower than occupational standards (e.g., ACGIH, NIOSH, and OSHA), and they include a margin of safety.

As discussed above, in the United States there are different standards of acceptable levels of contaminants set according to which population needs to be protected. The EPA's NAAQS, for the ubiquitous criteria pollutants, are targeted for the general population for continuous ambient exposures. ATSDR-MRLs give the concentrations of hazardous substances that will likely not affect the general population through daily exposure. NIOSH, OSHA, and ACGIH standards are for occupational exposures during 40-hour workweeks lasting the worker's lifetime.

The armed forces deployed to the Gulf theater are a unique segment of the population. They are predominately young (18–40), fit, and healthy. They stayed in the region after the onset of the Kuwait oil fires for (at most) 6–8 months. There are no studies in the scientific literature that focus on the possible health effects due to exposures to contaminants in this population segment. What standards should be considered appropriate for this group, exposed for 24 hours over a limited period of time? The NAAQS and MRL ambient levels are unrealistic because of the margins of safety included to protect the most vulnerable members of the population. The NIOSH, OSHA, and ACGIH occupational standards, although seemingly more appropriate, assume a step-function exposure with periods of low levels in-between but lasting for many years. Perhaps the occupational standards combined with an empirical factor to account for the continuous exposure would give a reasonable exposure-limit estimate.

MEASUREMENTS TAKEN BEFORE THE OIL WELL FIRES

Scant environmental data from the region exist to use as a baseline. Outdoor NO and NO_2 concentrations were measured at three sites in the city of Riyadh, Saudi Arabia (the Hitachi Building, at Al Matar Street; Al-Khazzan Street, one block from Makkah Road; King Saud University, Department of Civil Engineering), in 1986 and 1987. Table 2.4 illustrates the measurements from Al-Khazzan Street in 1987 (the location with the highest concentrations), and compares them with the NAAQS, the EPA standard, and the NIOSH occupational TWA values standards. The outdoor NO and NO_2 concentrations respectively are at least 270 and 14 times the reported average worldwide. Most of the outdoor NO and NO_2 in Riyadh was attributed to the 1,700,000 cars registered in 1984, in an area about 1900 km^2 with a population of 1,600,000 (Rowe, 1991).

Concentrations of NO_2 were measured throughout the state of Bahrain from January 6 to January 29, 1991, at the onset of the Persian Gulf War and before

Table 2.4

NO and NO_2 Concentrations in Riyadh, Saudi Arabia, in 1987 (ppm)

	Riyadh Concentration		EPA—NAAQS Ambient Standard		ACGIH Occupational Standard	
	NO	NO_2	NO	NO_2	NO	NO_2
Peak	9.2	1.48				
Maximum average hourly	2.7	0.16				
Maximum average daily	2.0	0.05				
Overall sampling average	0.86	0.040	NA	0.053	25	3.0

SOURCE: Rowe, 1991.
NOTE: NA = not applicable.

the burning of the oil wells. A large number (approximately 100,000) of jet flights took place during that period. The highest NO_2 levels were concentrated in Manama, the capital, with weekly mean values up to 0.145 ppm (273 $\mu g/m^3$), while the lowest were in coastal villages, which had means around 0.028 ppm (53 $\mu g/m^3$). Industrial areas had lower levels than the urban areas, which is characteristic where high traffic densities are the main source of NO_2 (Danish and Madany, 1992).

Even though only limited environmental data are available, high particulate levels have been documented throughout the Persian Gulf region: 5080 $\mu g/m^3$ in Riyadh, Saudi Arabia in 1982; 630 $\mu g/m^3$ in Kuwait in 1983; and 674 $\mu g/m^3$ in Bahrain in 1987 (Madany and Raveendran, 1992). A study from Dhahran, Saudi Arabia, reports TSP[1] measurements for 100 days from November 1980 to March 1981. The average maximum daily value for the period was 737 $\mu g/m^3$, and the geometric mean for the period was 339 $\mu g/m^3$. But when the Shammal winds were blowing, peak daily concentrations reached the extraordinary values of 2923 $\mu g/m^3$ (Khattak, 1982). These values are about 20 times the 24-hour U.S. ambient standard for PM_{10} of 150 $\mu g/m^3$. The daily variability in PM_{10} concentration is illustrated in Figure 4.2 for Doha, Kuwait, from June to December 1991.

MEASUREMENTS TAKEN DURING THE OIL WELL FIRES

No systematic environmental monitoring occurred in the Gulf region from the initial deployment in 1990 until May 1991. Several independent teams went to Kuwait to assess the ambient air contamination due to emissions produced by

[1]Total suspended particulate (TSP) is a historical term for airborne particles under about 30 μm. TSP has been replaced by the more precise and regulated parameter, PM_{10}, i.e., particulate matter of ≤10 μm.

the oil well fires. Measurements began in March 1991 when most of the fires were still burning. The most extensive measurement campaign was undertaken by the U.S. Army Environmental Health Agency (USAEHA) from May to December 1991. Other groups, like the EPA Team (Hunt, 1993), sampled for short periods; these provided only snapshots of the pollution levels, but they are still useful measurements for validating the more comprehensive data obtained by USAEHA.

May 6–June 12 Measurements

Airborne measurements of the oil-fire smoke were acquired by flights through the plumes from May 6 to June 12, 1991, while about 500 oil wells were still burning (Ferek, 1992). These measurements were taken from individual plumes (black and white), from composite plumes, and superplumes that originated from different fields as well as from a small number of pool fires. Table 2.5 summarizes the mean concentrations measured. In general, soot, salt, and sulfate account for about two-thirds of the total mass of smoke particles with diameters ≤ 3.5 μm.[2] Since data on only a few plumes were obtained, these results represent only a small portion of the entire episode; nevertheless, they are valuable additions to the body of information.

July–August Measurements

A study sponsored by the National Aeronautics and Space Administration (NASA) and the EPA consisted of plume and ground-level sampling. They measured the gas and particulate composition of the plumes and endeavored to assess their effect on the air quality in Kuwait City, in the region, and across the globe. This study was carried out during July and August 1991, when about half the fires were still burning.

The ground-level sampling in Kuwait City was taken during an abrupt change in weather conditions on August 7. Until August 5, winds with speeds of 35–55 km/hr (10–15 m/sec) transported the smoke from the oil fields toward the Persian Gulf, with little visual evidence of a plume across Kuwait City. On August 5, a strong, low-lying inversion developed, winds calmed, and visibility declined sharply. Winds then returned from the northwest, and, by August 9, dispersed the material that had accumulated over the city.

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[2]The cut-off value of 3.5 μm, $PM_{3.5}$, was arbitrarily set in the particle analyzer. These measurements were taken before the $PM_{2.5}$ standard was promulgated and is the closest available data above $PM_{2.5}$.

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Table 2.5

Mean Concentrations of Major Gases and Particulates from Different Types of Smoke Plumes from the Kuwait Oil Fires (in μg /m^3)

Plume Type & Date Sampled (1991)	CO_2	TOC[a]	CO	Soot (Elemental carbon)	CH_4	SO_2	NO_x	Particles <3.5 μm Diameter	Salts[c]
Super-composite plume (160 km downwind from Kuwait City) May 28	10,313 (98.1)[b]	92 (0.9)	52 (0.5)	36 (0.3)	20 (0.2)	215	4	241	50.5
Composite plume from Greater Burgan field (20 km downwind of fires) June 12	29,143 (95.1)	915 (3.0)	264 (0.9)	224 (0.7)	99 (0.3)	423	25	910	2819
Individual black plume (in Umm Qudair field) June 9	8,107 (90.7)	599 (6.7)	151 (1.7)	58 (0.6)	19 (0.2)	455	4	166	4.9
Individual white plume (in north field) June 8	35,893 (99.2)	118 (0.3)	137 (0.4)	33 (0.1)	15 (0.04)	1041	29	1,093	880
From a pool fire (in Minagish field) June 2	15,536 (97.1)	37 (0.2)	106 (0.7)	308 (1.9)	13 (0.1)	1326	16	790	0.8

SOURCE: Ferek, 1992.

[a]TOC = Total nonmethane organic carbon in the vapor phase. [b]Percentage of carbon specie contributing to the total carbon in the plume is included in the parentheses. It indicates combustion efficiency; a higher percentage means more efficient combustion. [c]Salts = Sum of all salts measured.

In the August 7 sample, the dominant component was sand dust. Sulfur, as SO_2, increased by about 50 percent during the inversion. Concentrations of Cl, Pb, Br, and Zn increased at least ten-fold. The ratio of Pb to Br of about three remained constant throughout the sampling, suggesting that their primary source was local automotive traffic and not the oil well fires. Concentrations of Ca, K, Ti, Fe, and Mn increased by less than 50 percent during the inversion period. The ratios of Si, Fe, K, Ti, and Mn to Al were within 30 percent of the values for local desert soils and similar to soils in Arizona. PAH levels in Kuwait City were typically less than 0.001 $\mu g/m^3$ as shown in Table 2.11 (Stevens et al., 1993).

July 31–August 4 Measurements

A five-day study was performed in Bahrain from July 31 to August 4, 1991, collecting particulate matter (PM_{10}) that was also analyzed for 31 polycyclic aromatic compounds (PAH), nickel, and vanadium (Madany, 1992). During the data-acquisition period, temperatures ranged from 35° to 38°C; winds ranged from 11 to 31 km/hr; the height of the inversion layer ranged from 457 to 914 m; the air was smoky; and all sampling filters appeared black.

The daily PM_{10} concentrations ranged from 139 to 673 $\mu g/m^3$ but, although quite high, cannot be attributed only to the oil fires. Comparison with available data on particulate matter from before the Gulf War shows that these levels are common for the region.

The mean daily concentrations of Ni and V ranged from 7 to 42 ng/m^3 and 11 to 42 ng/m^3, respectively. They were strongly correlated, indicating that they had a common source: the oil fires (Madany, 1992).

Table 2.6 depicts the average concentrations of individual components of the PAHs in Bahrain for the study period as well as what is considered background, rural, and urban values for cities around the world for comparison (Madany, 1992). It is important to note that the PAH levels in Bahrain, which is downwind from Kuwait, were at least an order of magnitude lower than the European cities with high PAH concentrations.

A study on a "positive" aspect of the Kuwait oil fires looked into the comfort of the residents of Jubail, Saudi Arabia, during the Kuwait oil fires. Many Jubail residents said that it had been noticeably cooler since the fires were ignited in Kuwait. Some residents speculated that temperatures were 10° to 20°C below normal. Comparison of the mean air temperatures for the months of May and June 1991 with historical data for the same months indicates that the actual decrease in temperatures was not statistically significant. On the other hand,

Table 2.6

Levels of PAHs Measured in Bahrain and Other Sites (ng/m^3)

Compound	Alaska	Lake Superior	Netherlands		Greece	Berlin	San Francisco	Bahrain
	Background		Rural	Urban	Urban	Urban		
Anthracene	0.01	0.01	0.9	5.4	NA	NA	NA	0.04
Fluoranthene	0.16	0.10	11.0	39.0	3.6	33.0	NA	0.09
Benzo(a)anthracene	NA	NA	0.5	1.3	3.6	NA	NA	0.07
Crysene	0.04	0.06	1.5	3.1	3.7	NA	NA	0.20
Benzo(k)fluoranthene	NA	NA	0.5	0.7	NA	4.2	0.2	0.14
Benzo(a)pyrene	0.02	0.04	0.5	0.6	6.7	9.1	0.4	0.62
Benzo(g,h,i)perylene	0.04	0.04	0.9	1.2	8.0	11.0	1.4	0.85
Ideno(1,2,3-cd)pyrene	NA	NA	0.8	1.0	NA	8.2	NA	0.59

SOURCE: Madany, 1992.

NOTE: NA = not available.

the mean temperatures for January to April were slightly higher than those predicted from historical data. Solar radiation data indicate a 26–36 percent decrease compared to the same months during 1979–1990. According to one researcher (Riley, 1992), "One way of looking at these results is to think of the smoke plumes as being analogous to the shade of an extended palm grove. The air temperature in the shade is the same as in full sunlight, but the level of human comfort is significantly higher under the trees because the solar-radiation load on an individual is reduced." This is an example of how the general population's perception of an effect can lead to a wrong conclusion.

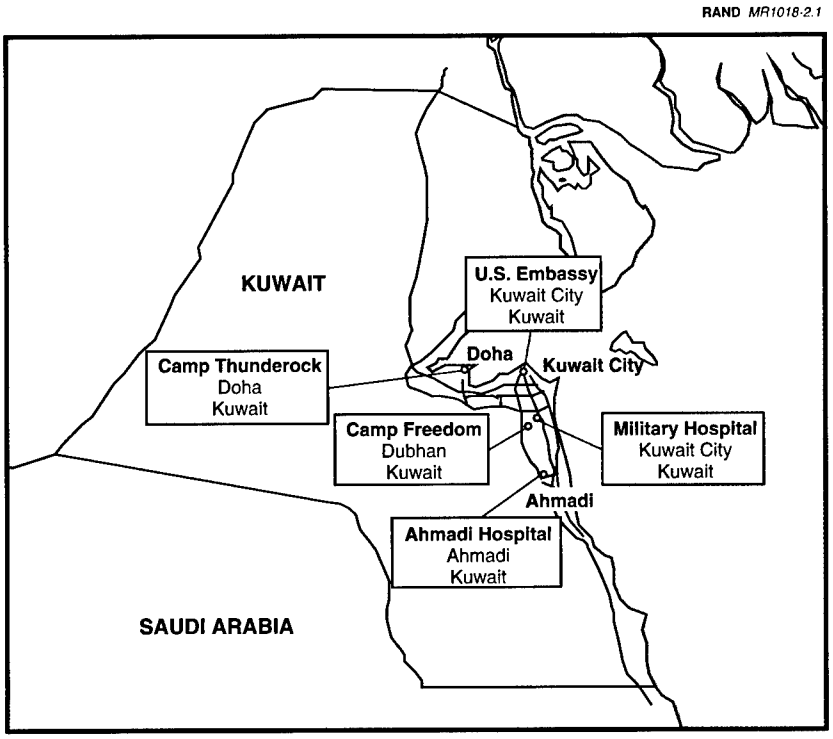
USAEHA Environmental Measurements, May–December

The USAEHA, renamed the U.S. Army Center for Health Promotion and Preventive Medicine (CHPPM) in 1991, was commissioned to determine whether dangerous levels of pollutants from oil-fire emissions were present in U.S. troop locations. Also, in accordance with Public Law 102-190, CHPPM was to determine the potential health risks to DoD personnel deployed in Operation Desert Storm from exposures to those pollutants. Environmental monitoring began on May 15, 1991, and continued until December 3, 1991, with about 4000 samples collected. When data collection began, 558 oil wells were burning—only 8 percent of the fires had been extinguished. The last fire was extinguished on November 6, 1991.

Permanent ambient air monitoring stations were established at four locations in Saudi Arabia and six in Kuwait, although two in Kuwait had to be abandoned quickly due to logistical difficulties (see Figures 2.1 and 2.2). The sites selected were locations where large concentrations of U.S. troops and DoD civilians were stationed for long periods of time. The two mostly civilian sites were the U.S. Embassy in Kuwait and the Al Ahmadi Hospital; the others were mostly military sites.

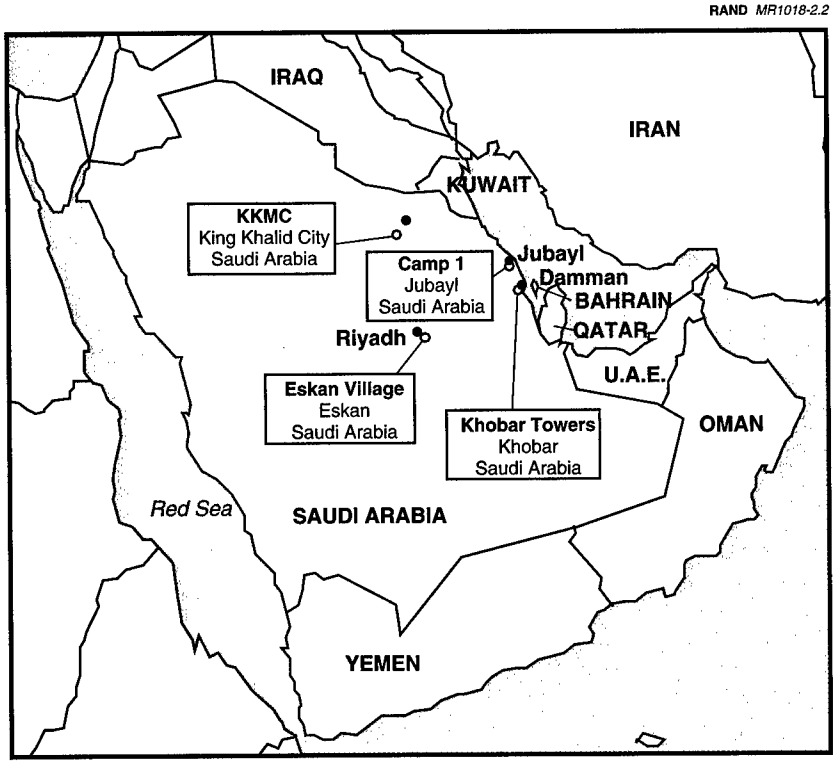
Based on the hazardous substances to be monitored—crude oil, by-products of incomplete combustion, breakdown products—scientists from USAEHA decided on a list of pollutants of concern that needed to be monitored in the Gulf region affected by the oil well fires (Table 2.7).

Soil Sampling. Soil sampling was also performed at the air sites. There were no consistent increases in soil metals concentrations above background, except for lead. Lead increased at all sites, as it did in the air data, probably reflecting an increase in vehicular traffic and subsequent lead emissions from local gasoline. There were no VOC or PAH levels above the instrumental detection limit. There also was no detection of Cr(VI) in 76 samples analyzed with a detection limit of 100 ppb (USAEHA, 1994).



SOURCE: USAEHA 1994.

Figure 2.1—Monitoring Sites in Kuwait



SOURCE: USAEHA 1994.

Figure 2.2—Monitoring Sites in Saudi Arabia

Air Sampling. The USAEHA sampling campaign measured little change in the general air quality during the monitoring period. Table 2.8 lists the highest mean values of the contaminants listed in Table 2.7 as measured by the USAEHA during their 1991 campaign. Although considerable increases were noted in particulate matter, these concentrations were considered to be within the range common to this area.

Exposures to several VOCs (i.e., benzene, toluene, ethyl-benzene, and xylene) were similar to levels observed in cities with major petrochemical industries (i.e., Houston and Philadelphia (USAEHA, 1994)). Figure 2.3 compares median VOC concentrations in Kuwait, Saudi Arabia, and several U.S. cities. The median VOC concentrations for benzene, toluene, ethyl-benzene, and the xylenes at the Kuwait and Saudi Arabian sites are comparable to concentrations in ur-

Table 2.7

List of the Pollutants Monitored by USAEHA

Volatile Organic Compounds (VOC)		
Benzene	o-Xylene	Heptane
Toluene	p-Xylene	
m-Xylene	Ethyl benzene	
Polycyclic Aromatic Hydrocarbons (PAH)		
Acenaphthene	Benzo (g,h,i) perylene	Ideno(1,2,3 -cd)pyrene
Acenaphthylene	Biphenyls	Methylnaphthalene
Anthracene	Carbazole	Naphthalene
Benzo(a)anthracene	Chrysene	Phenanthrene
Benzo(a)pyrene	Cumene	Pyrene
Benzo(b)fluoranthene	Dibenzo(a,h)anthracene	1-Methylnaphthalene
Benzo(e)pyrene	Dibenzofurans	2,6-Dimethylnaphthalene
Benzo(f)fluoranthene	Fluoranthene	2-Methylnaphthalene
Acidic Gases		
Hydrochloric acid	Nitric acid	Sulfuric acid
Criteria Pollutant Gases		
Nitrogen dioxide	Nitric oxide	Ozone
Sulfur dioxide		
Particulates and Metals		
PM_{10}	Nitrates	Sulfates
Aluminum	Chlorine	Mercury
Arsenic	Chromium	Nickel
Beryllium	Iron	Sodium
Cadmium	Lead	Vanadium
Calcium	Magnesium	Zinc

SOURCE: USAEHA, 1994.

Table 2.8

Mean Concentrations of Air Pollutants of Concern in May–December 1991

Pollutant		Location	Mean [c][a]	ACGIH's TLVs
Ozone	O_3	Riyadh	53.4 µg/m^3	100[c] µg/m^3
Sulfur dioxide	SO_2	Riyadh	23.8 µg/m^3	5200 µg/m^3
Nitrogen dioxide	NO_2	Khobar	58.5 µg/m^3	5600 µg/m^3
Nitric oxide	NO	Khobar	24.2 µg/m^3	31,000 µg/m^3
Acenaphthene[b]		Eskan Village	0.62 ng/m^3	200,000 ng/m^3
Benzo(a)anthracene[b]		Eskan Village	0.60 ng/m^3	200,000 ng/m^3
Biphenyl[b]		Eskan Village	7.20 ng/m^3	200,000 ng/m^3
Chrysene[b]		Eskan Village	0.48 ng/m^3	200,000 ng/m^3
Fluoranthene[b]		Eskan Village	1.41 ng/m^3	200,000 ng/m^3
Phenanthrene[b]		Ahmadi	0.48 ng/m^3	200,000 ng/m^3
Pyrene[b]		Eskan Village	0.65 ng/m^3	200,000 ng/m^3
Particulate	PM_{10}	Ahmadi	354 µg/m^3	3000[d] µg/m^3
Cadmium	Cd	Camp 1	0.003 µg/m^3	10 µg/m^3
Chromium III	Cr	Camp 1	0.027 µg/m^3	500 µg/m^3
Nickel	Ni	U.S. Embassy	0.052 µg/m^3	120 µg/m^3
Lead	Pb	Eskan Village	0.675 µg/m^3	50 µg/m^3
Vanadium	V	Ahmadi	0.028 µg/m^3	50 µg/m^3
Zinc	Zn	Camp 1	0.068 µg/m^3	500 µg/m^3
Benzene		Ahmadi	7.82 µg/m^3	1600 µg/m^3
Toluene		Ahmadi	21.8 µg/m^3	188,000 µg/m^3
Ethyl benzene		Ahmadi	14.7 µg/m^3	435,000 µg/m^3
m,p-Xylene		Ahmadi	40.5 µg/m^3	435,000 µg/m^3
0-Xylene		Ahmadi	12.8 µg/m^3	435,000 µg/m^3

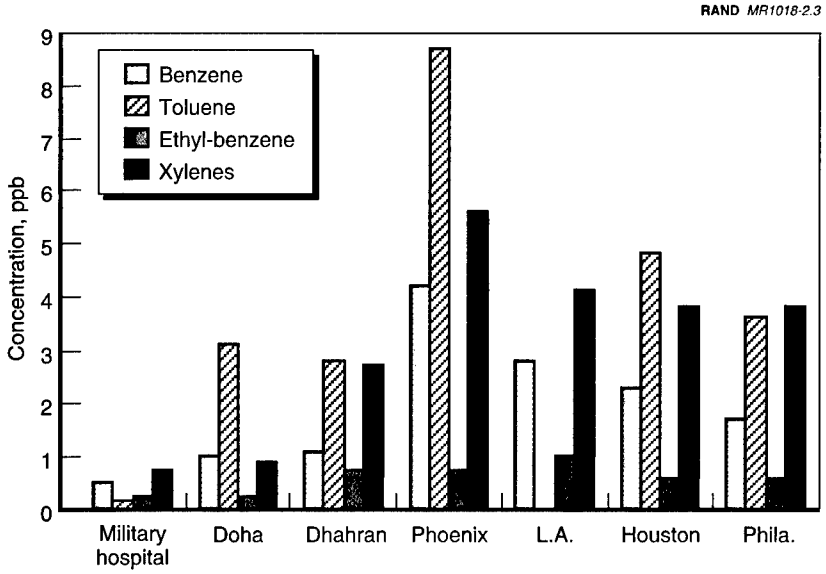
NOTES: NA: Standard or recommended concentration not available.

[a]USAEHA, 1994. [b]There were only a few samples above the detection limit. [c]TLV performing heavy work. [d]PM_{10} not otherwise regulated.

ban centers in the U.S. The levels of NO_2, CO, SO_2, H_2S, and PAHs were lower than expected, given the magnitude of the fires, and did not exceed those seen in U.S. cities or the EPA standards where established.

High levels of airborne particulate matter (sand and soot) were observed at several monitoring sites. Analysis of the samples indicated that the particles were mostly sand-based materials; high levels of airborne sand particulates are typical for this region of the world (Kirkpatrick, 1997). Within the PM_{10} samples of particulate matter, levels of PAHs and toxic metals were low.

A small subset of the ambient air samples collected in Kuwait and Saudi Arabia were analyzed to determine particle-type class and particle-size distribution of the PM_{10} data. The assigned classes fell into the following main categories: earth crustal (silica-rich, e.g., quartz), calcium bearing (calcium-rich, e.g.,



SOURCE: USAEHA, 1994.
NOTE: Toluene measurement for Los Angeles not available.

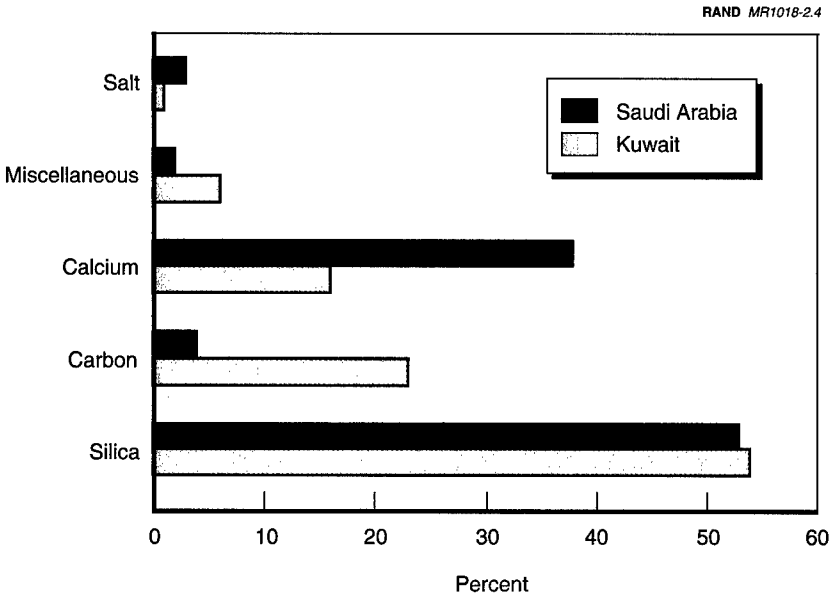
Figure 2.3—Median VOCs Comparisons: Kuwait, Saudi Arabia, and Selected U.S. Cities

dolomite, gypsum), salt particles, carbon rich (e.g., soot), and miscellaneous (USAEHA, 1992). Figure 2.4 shows the percentile composition of the particulate matter in the air of Kuwait and Saudi Arabia (USAEHA, 1992). Considering that the sand of the Arabian Peninsula is rich in calcium and silica, it indicates that most of the PM_{10} is of sand origin and that in Kuwait about 23 percent of the total PM_{10} mass was contributed by soot from the oil well fires.

Figure 2.5 depicts the particle-size distribution in air samples from Kuwait and Saudi Arabia. The results from a small number of samples indicate a significant mass of particles in the PM_{10} range.

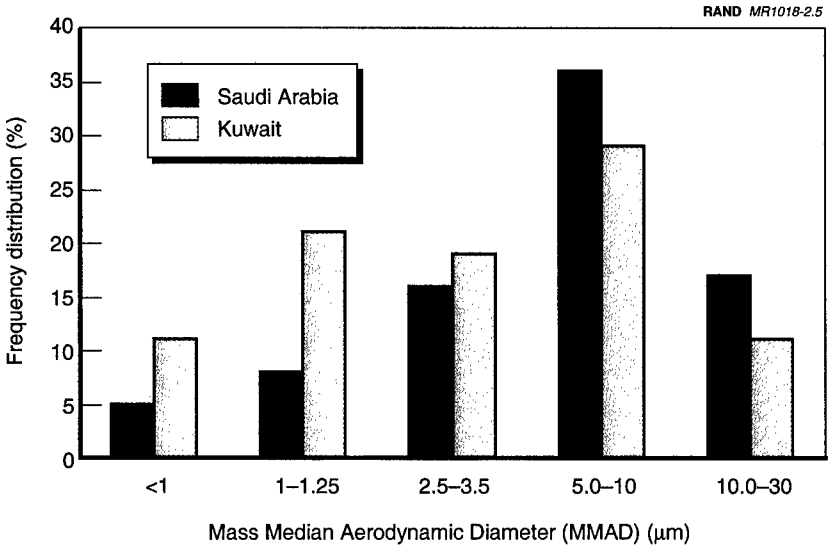
USAEHA Industrial Hygiene Measurements, May–June

A team of industrial hygienists from USAEHA performed supplemental monitoring at various sites in Kuwait and Saudi Arabia from May 3 to June 17, 1991. The focus of this operation was to sample areas where DoD service members were working outdoors at possible worst-case locations within the oil fields next to Kuwait City. Measurements focused on Khobar Towers, where large numbers of troops awaited departure from the region through the ports of Dammam and Jubayl, and on Camp Thunderock in Kuwait City. At the time of this assessment, DoD personnel were neither working nor living in the vicinity of the



SOURCES: Kirkpatrick, 1997; USAEHA, 1992.

Figure 2.4—Particulate Composition of Air Samples in Saudi Arabia and Kuwait



SOURCES: Kirkpatrick, 1997; USAEHA, 1992.

Figure 2.5—Particle Size Distribution, Kuwait and Saudi Arabia

oil fires. The workers in the oil fields were employees of fire fighting companies. These measurements are equivalent to personal sampling to determine occupational exposures while performing specific jobs or operations. The operations, all outdoors, included guard posts, supply points, equipment handling sites, ammunition handling and storage sites, and residential and field work locations.

The results are summarized in Table 2.9. The table provides means and standard deviations, maximums measured, and ACGIH's TLVs. Most measured pollutant concentrations were well below the occupational TLVs.

MEASUREMENTS TAKEN AFTER THE OIL WELL FIRES

The USAEHA conducted an additional monitoring effort in November 1993 in Camp Thunderock, Doha, Kuwait, and Khobar Towers, Dhahran, Saudi Arabia, to provide additional information on postfire air pollution levels. The mean concentrations of the contaminants of interests for Camp Thunderock and Khobar Towers measured during November 1991 and November 1993 are listed in Table 2.10. The 1993 campaign was a limited effort, and these means should be considered only as an indicator of air pollution levels.

The results indicate that the metals and VOC readings at Khobar Towers in 1993 were about the same as those in 1991, but PAH and PM_{10} levels were lower. The November 1991 VOC data for Khobar Towers are similar to the November 1993 levels, except for toluene. The Khobar Towers VOC levels are a factor of three higher than Camp Thunderock. The PAH levels detected in November 1993

Table 2.9

Summary of Industrial Hygiene Air Sampling in Kuwait and Saudi Arabia (mg/m^3)

Pollutant	Kuwait[a]	Saudi Arabia[a]	Maximum Value	ACGIH's TLVs
PM_{10}	0.35 (0.64)	0.46 (0.24)	2.00	3.0
Coal Tar Pitch	0.06 (0.96)	0.10 (0.09)	0.21	0.2
Nitrogen Dioxide	1.65 (1.30)	0.40 (0.24)	6.70	5.6
Sulfur Dioxide	0.47 (0.72)	0.53 (0.65)	1.90	5.2
Nitric Acid	0.05 (0.02)	0.04 (0.05)	0.12	5.2
Sulfuric Acid	0.04 (0.01)	0.03 (0.01)	0.05	1.0
Benzene	0.04 (0.01)	BDL	0.19	1.6
Toluene	0.13 (0.06)	BDL	0.75	188
Xylene	0.37 (0.08)	BDL	3.21	435

SOURCE: USAEHA, 1994; ACGIH, 1997.

NOTES: [a]mean (standard deviation). BDL: Below Detection Limit.

Table 2.10

Comparison of 1991 and 1993 Mean Contaminant Concentrations ($\mu g/m^3$)

	Khobar Towers		Camp Thunderock	
Contaminant	1991	1993	1991	1993
PAHs				
Naphthalene	1.10	0.11	2.14	0.05
2-Methylnaphthalene	0.21	0.05	0.24	0.02
Dibenzofuran	0.17	0.004	0.13	NA
Fluorene	0.03	0.004	0.1	NA
Phenanthrene	0.04	0.008	0.02	0.02
PM_{10}	62	52	84	44
Metals				
Cadmium	0.001	0.0009	0.001	0.0002
Chromium	0.003	0.013	0.017	0.005
Nickel	0.02	0.007	0.05	0.02
Lead	0.29	0.35	0.26	0.1
Vanadium	0.005	0.005	0.007	0.005
Zinc	0.05	0.05	0.07	0.03
VOCs				
Benzene	4.5	3.4	10.6	1.2
Toluene	18.5	8.9	33.9	2.7
Ethyl Benzene	3.9	2.7	3.6	1.0
m,p-Xylene	11.3	6.1	18.8	2.7
o-Xylene	4.3	2.9	17.1	1.2

SOURCES: 1991 data: USAEHA, 1994; 1993 data: Kirkpatrick, 1997.
NOTE: NA = Not available.

were 2 to 8 times lower than in November 1991. For PM_{10} the November 1993 mean levels are lower than in November 1991, decreasing from a mean concentration of 62 $\mu g/m^3$ to 52 $\mu g/m^3$.

At Camp Thunderock, the readings for all four categories were substantially lower in 1993 than they were in 1991. The metal levels from November 1993 are a factor of 2 lower than those in November 1991. In the November 1993 PAH data, most of the compounds are below detection limit and the rest are about three times lower than those in November 1991. PM_{10} levels decrease from 84 $\mu g/m^3$ to 44 $\mu g/m^3$.

CONCLUSIONS

This chapter summarizes the available data on environmental contaminants measured in Kuwait and Saudi Arabia beginning when most of the oil wells were still burning and ending after the fires were extinguished. U.S. occupational guidelines for contaminants offer a practical standard against which to

measure exposure levels experienced by U.S. troops. As discussed above, ACGIH standards are set for workplace exposures for 8-hour days and 40-hour weeks, or about 2000 hours per year for a typical working career of 40–45 years.

Table 2.11 displays the maximum or 95th-percentile concentrations in the air for the pollutants of concern as measured by the USAEHA from May to December 1991, and the ACGIH-recommended exposure limits for hazardous substances in the workplace. These results show that, except for ozone, the maximum concentrations for all pollutants in the Gulf were several orders of magnitude lower than ACGIH occupational standards. And ozone, which is ubiquitous in the U.S., was still lower than in many U.S. urban areas where summer episodes reach twice the NAAQS.

A possible objection to the above comparison is that U.S. personnel in the Gulf were being exposed to air pollution 168 hours per week for up to 70 weeks, while occupational exposures are spread over 40 hours per week during an adult working lifetime. Nevertheless, if the mean pollutant concentrations due to the Kuwait oil well fires are compared to the NAAQS and to the ATSDR's MRL levels, the Gulf levels were lower than the U.S. standards set for the general population (except for PM_{10}), and lower than the daily exposures of millions living in major U.S. cities. However, PM_{10} levels, unrelated to the oil well fires, were much higher than ambient levels in the U.S., but within the ACGIH's TLV levels. Gulf War veterans, like people living in urban areas in the U.S., were exposed to multiple pollutants simultaneously for which there are no standards.

Table 2.11

Maximum, or 95th Percentile, Concentrations of Air Pollutants of Concern

Pollutant	Sym	Location	ACGIH TLVs	Maximum [C][a]	Maximum [C][b]
Ozone	O_3	Camp Thunderock	100 µg/m^3	104.8 µg/m^3	(Kuwait City only)
Sulfur dioxide	SO_2	Ahmadi	5200 µg/m^3	92.5 µg/m^3	11.0 µg/m^3
Nitrogen dioxide	NO_2	Khobar	5600 µg/m^3	86.1 µg/m^3	
Nitric oxide	NO	Khobar	31,000 µg/m^3	61.1 µg/m^3	
Acenaphthene[c]		Eskan Village	200,000 ng/m^3	2.25 ng/m^3	<1 ng/m^3
Benzo(a)anthracene[c]		Eskan Village	200,000 ng/m^3	2.23 ng/m^3	<1 ng/m^3
Biphenyl[c]		Eskan Village	200,000 ng/m^3	19.07 ng/m^3	<1 ng/m^3
Chrysene[c]		Eskan Village	200,000 ng/m^3	2.25 ng/m^3	<1 ng/m^3
Fluoranthene[c]		KKMC	200,000 ng/m^3	2.23 ng/m^3	<1 ng/m^3
Phenanthrene[c]		Ahmadi	200,000 ng/m^3	1.84 ng/m^3	<1 ng/m^3
Pyrene[c]		Eskan Village	200,000 ng/m^3	3.54 ng/m^3	<1 ng/m^3
Particulate	PM_{10}	U.S. Embassy	3000 µg/m^3	1842 µg/m^3	
Cadmium	Cd	Camp 1	10 µg/m^3	0.0078 µg/m^3	
Chromium	Cr	U.S. Embassy	500 µg/m^3	0.0898 µg/m^3	0.013 µg/m^3
Lead	Pb	Eskan Village	50 µg/m^3	1.596 µg/m^3	1.671 µg/m^3
Nickel	Ni	Camp 1	120 µg/m^3	0.2136 µg/m^3	0.0081 µg/m^3
Vanadium	V	Camp 1	50 µg/m^3	0.0898 µg/m^3	0.0093 µg/m^3
Zinc	Zn	Camp 1	500 µg/m^3	0.193 µg/m^3	0.172 µg/m^3

Table 2.11—continued

Pollutant	Sym	Location	ACGIH TLVs	Maximum [C][a]	Maximum [C][b]
Benzene		Ahmadi	1600 $\mu g/m^3$	13.1 $\mu g/m^3$	
Toluene		Ahmadi	188,000 $\mu g/m^3$	36.9 $\mu g/m^3$	
Ethyl benzene		Ahmadi	435,000 $\mu g/m^3$	41.2 $\mu g/m^3$	
m,p-Xylene		Ahmadi	435,000 $\mu g/m^3$	116 $\mu g/m^3$	
o-Xylene		Ahmadi	435,000 $\mu g/m^3$	30.4 $\mu g/m^3$	

[a]For particulates, metals, and VOCs, this column gives the 95th percentile [C]; for PAHs and Criteria Pollutants it gives the maximum [C] (USAEHA, 1994). [b]Stevens et al., 1993. [c]There were only a few samples above the detection limit. The ACGIH TLV is for coal tar pitch volatiles.

Chapter Three

POSSIBLE HEALTH EFFECTS OF OIL FIRES

This chapter describes the possible health effects due to exposures to the pollutants of concern. The information and conclusions that follow are based on key primary sources, i.e., publications in peer-reviewed scientific journals. The information presented here has been restricted to the range of exposures of interest that are based on the maximum concentrations measured in the region as depicted in Table 2.11. The section describes what is known about the health effects of VOC, PAH, particulate matter, acidic aerosols, metals, criteria and photochemical pollutants, and long-term exposures to air pollution. The last section summarizes the results of several health studies related to Gulf War veterans conducted during and after the conflict. It also includes an overview of the symptoms reported by Gulf War veterans to complete the stand-alone report. Again, it in no way purports to be either a critical or comprehensive review.

VOLATILE ORGANIC COMPOUNDS

Benzene

Benzene is present, at low levels, in many plants and animals. Although natural sources such as volcanoes and forest fires emit small amounts of benzene, the major releases arise from the use of crude oil and gasoline, and from chemical-industry emissions. The most common sources of benzene exposure for humans are gasoline filling stations, tobacco smoke, and vehicle exhaust fumes. Because benzene is very volatile, the prevalent form of exposure is by inhalation, followed by ingestion of contaminated foods and water, and last by dermal contact, mainly with products containing benzene such as gasoline.

For the general population, benzene standards are set by the EPA for drinking water at 5 ppb (1 ppb = 3.20 $\mu g/m^3$), with the ultimate goal of 0 ppb in drinking water and in lakes and rivers. NIOSH's recommended occupational TWA is 100 ppb, OSHA's TWA is 1000 ppb, and ACGIH's TLV is 500 ppb.

Table 3.1

Benzene Concentrations in Outdoor Air (ppb)

Locations	Mean Concentration Range	Comments	References
Denver, CO	24.5 17.9–39.5	Urban, summer 1986	EPA, 1987
73 km NW of Denver, CO	0.02–0.85	Rural, spring–fall 1982	Roberts, 1985
Manhattan, NY	10.5 5.3–31.8	Urban, summer 1986	EPA, 1987
Staten Island, NY	6.6 0.1–34	Urban, spring 1984	Singh, 1985
Elizabeth & Bayonne, NJ	3.0 max. 13.8	Urban, daytime, fall 1981	Wallace, 1985
Elizabeth & Bayonne, NJ	2.7 max. 28.5	Urban, at night, fall 1981	Wallace, 1985
Chicago, IL	20.7 3.8–30.3	Urban, summer 1986	EPA, 1987
St. Louis, MO	11.1 3.8–2.7	Urban, summer 1985	EPA, 1987
Stinson Beach, CA	0.38 ± 0.39	Remote coastal, 1984	Wester, 1986
Al Ahmadi Hospital	4.2 Maximum		USAEHA, 1994

Benzene is ubiquitous in air, both in rural and urban areas as well as indoors. Table 3.1 lists benzene concentrations in several U.S. locations and at the Al Ahmadi hospital.

Table 3.2

Daily Median Air Benzene Concentrations (ppb)

Location	Concentration
Remote	0.16
Rural	0.47
Suburban	1.8
Urban	1.8
Indoor air	1.8
Workplace air[a]	2.1

SOURCE: Shah, 1988.

[a]These measurements were made prior to current restrictions on smoking in the workplace. Current levels are most likely lower.

Table 3.3

Benzene Concentrations in Air at Different Sites (ppb)

Site	Concentration	Reference
Inside home w/0 smokers	2.2	Wallace, 1989a
Inside home w/1 or more smokers	3.3	Wallace, 1989a
Inside a smoke-filled bar	8.1–11.3	Brunnemann, 1989
Breath of smokers	4.7	Wallace, 1989b
Breath of nonsmokers	0.47–0.63	Wallace, 1989b
Breath of smokers in urban area	6.8±3.0	Wester, 1986
Breath of nonsmokers in urban area	2.5 ± 0.8	Wester, 1986
Breath of smokers in remote area	2.1 ± 9.6	Wester, 1986
Breath of nonsmokers in remote area	1.8 ± 0.2	Wester, 1986
In vehicle on NJ Turnpike	5.0 ± 6.0	Lawryk, 1995
In vehicle in Lincoln Tunnel, NY–NJ	8.1 ± 8.3	Lawryk, 1995
Municipal landfill	32	Wood, 1987
Kin-Buc Landfill, Edison, NJ (Superfund site)	59.5	Bennett, 1987
Love Canal basement, Niagara Falls, NY	162.8	Pellizzari, 1982

Daily median benzene air concentrations for the period 1975–1985 are listed in Table 3.2. The outdoor air values include data from 300 cities in 42 states and the indoor air from 30 sites in 16 states (Shah, 1988).

Personal exposures[1] to benzene tend to exceed the outdoor air concentrations. Data from the Total Exposure Assessment Methodology (TEAM) study (Wallace, 1989a) give a mean personal exposure of about 4.7 ppb, compared to a mean outdoor concentration of only 1.9 ppb. The same study also measured the median level of benzene inside homes without smokers and with one or more smokers. The results are included in Table 3.3. It is interesting to note that the personal level is higher than the level inside the home. A plausible explanation is that the personal value also includes exposures at other locations where daily activities of the study volunteer take him or her, i.e., in transit, inside a car, at work, etc.

Benzene intake during daily activities can be estimated. A smoker who consumes approximately two packs per day will have an additional daily intake of about 1200 μg of benzene (Fishbein, 1992). Assuming an urban concentration range of 2.8–20 ppb and an air intake of 20m^3 per day, then the average air intake of benzene is 180–1300 μg per day. For a moving automobile with an average benzene concentration of 40 μg/m^3 and an exposure duration of one hour per day, the benzene intake would be approximately 40 μg per day (Wallace,

[1]Personal exposure is the concentration inhaled by a subject. It can be measured directly in the air around the mouth-nose of a subject, e.g., with a sampler attached to the lapel.

1989a). Estimates of exposure from self-filling a car with gasoline and from evaporative emissions seeping into a home from automobiles in attached garages have been set at 150 μg per day (Wallace, 1989a).

Human studies show that inhalation exposure to benzene in the 1,000-ppb range (1 ppm = 1,000 ppb) from several months to several years reduces the number of all major blood cell types—erythrocytes (red blood cells), platelets, and leukocytes (white blood cells)—that are produced in the bone marrow. The next stage of severity is aplastic anemia, when the bone marrow ceases to function. Aplastic anemia can progress to acute myelogenous leukemia. Several occupational studies of workers exposed to low levels of benzene (approximately 25,000 ppb for nine years (Fishbeck, 1978) and 2,000–35,000 ppb (Townsend, 1978)) indicate a slight decrease in red blood count (RBC) at the end of the exposure period, but normal values years later. Significant decreases in white- and red-cell counts were recorded for workers exposed to 75,000 ppb for 10 years; for later years at lower exposures (15,000–20,000 ppb) their blood counts increased to normal values (Kipen, 1989). More severe effects, such as preleukemia or acute leukemia, were observed in workers exposed to 210,000–650,000 ppb for 1–15 years (Aksoy, 1978). Painters exposed to 3,000–7,000 ppb of benzene and other VOCs for 1–21 years showed increased serum immunoglobin (IgM) values and decreased values of IgG and IgA (Lange, 1973).

Multiple animal studies support the above observations (Rozen, 1984, 1985). Animal studies indicate a decrease in functional immune responses reflected in decreased resistance to infectious agents at benzene concentrations ≥ 30,000 ppb for five days with recovery on the seventh day (Rosenthal, 1985).

The genotoxicity[2] of benzene has been studied extensively. Benzene and its metabolites seem to be genotoxic to humans, causing primarily chromosomal aberrations (Major, 1992; Yardley-Jones, 1990; Sasiadek, 1989).

Experimental data for animals and studies of humans indicate a link between a decrease in bone-marrow cellularity and the development of leukemia. Many cases of benzene-induced leukemia seem to have been preceded by aplastic anemia (Toft, 1982). Benzene is considered a human carcinogen by U.S. and international agencies; the EPA classifies it as a Class A variety. The EPA estimated a risk value of 2.7×10^{-2} for leukemia due to a total lifetime exposure of 1,000 ppb inhaled benzene for concentrations in air below 31 ppb (EPA, 1986).

[2]Genotoxic: denotes a substance that may cause mutation or cancer by damaging DNA.

Toluene

Toluene, a very volatile liquid, occurs naturally in petroleum crude oil and in the tolu tree (hence its name). The most preponderant sources of toluene are evaporation from gasoline and release through car exhaust. Levels of toluene in the outdoor environment range from 0.26–7.8 ppb (1-30 $\mu g/m^3$) in suburban and urban air (1 ppm = 3.75 $\mu g/m^3$) (Chan, 1991). However, the maximum air concentrations of toluene are found indoors in the range of 9.9–18.5 ppb (38–71 $\mu g/m^3$) (Wallace, 1991). This increased concentration arises from the use of household products such as paint, thinners, and glues; attached garages and cigarette smoking are also major household contributors.

The Clean Air Act Amendments of 1990 list toluene as a hazardous air pollutant. OSHA's TWA is 200 ppm (= 200,000 ppb = 750,000 $\mu g/m^3$); NIOSH's TWA is 100 ppm (= 100,000 ppb = 375,000 $\mu g/m^3$); and ACGIH's TLV is 50 ppm (50,000 ppb = 188,000 $\mu g/m^3$).

Studies of humans and animals have demonstrated that toluene is readily absorbed via the lungs and the gastrointestinal tract; it accumulates in adipose tissues (EPA, 1984). Elimination of toluene is primarily (approximately 2/3) via urine as hippuric acid and is usually complete within 24 hours of exposure. Low and intermediate levels of exposure to toluene primarily affect the central nervous system as summarized in Table 3.4 (Benignus, 1981). Effects were reversible, even at high-exposure levels for long durations.

Table 3.4

Health Effects of Toluene Exposure in Humans

Concentration	Duration	Symptoms	Reference
100 ppm[a]	4 days @ 6 hours per day	Headaches, dizziness, and eye irritation	Andersen, 1983
600 ppm	8 hours	All of the above plus euphoria, dilated pupils, convulsion and nausea	Benignus, 1981
200–800 ppm	chronic	All of the above plus fatigue, muscular weakness, confusion, and accommodation disturbances	Greenberg, 1997; Boey, 1997
10,000–30,000 ppm		Narcosis and death	Echeverria, 1989

[a]ppm = 1,000 ppb.

Epidemiological studies revealed no significant increased risk for cancer among workers, but toluene may produce liver and kidney damage at high levels of exposure (Benignus, 1981).

Xylene

Xylene occurs naturally in petroleum and coal tar, and is also formed during forest fires. It is a compound primarily used as a "safe" substitute for benzene, and is found in gasoline as part of the BTX component (benzene-toluene-xylene). Xylene has wide industrial use as a solvent and in the manufacture of synthetic agents.

Very little is known of its human health hazards compared to benzene and toluene, particularly chronic effects. Current animal data about whether xylene causes cancer are inconclusive. Epidemiological studies on cancer risks associated with toluene and xylene have to control for the known effects of benzene impurities (McMichael, 1988). Low levels (100–300 ppm) of inhaled xylene can cause eye, nose, and throat irritation, delayed response to visual stimuli, and reduced memory (Fishbein, 1985). NIOSH's and OSHA's TWA as well as ACGIH's TLV for m,o,p-xylene is 100 ppm. Exposure of workers in China to a mixture of toluene and xylene indicates that the effects of the combined toxicities are additive (Chen, 1994).

POLYCYCLIC AROMATIC HYDROCARBONS

PAHs are produced by incomplete combustion of coal, oil, gas, forest vegetation or other organic substances. Although only a few PAHs have limited commercial use, they are found throughout the environment in air, water, and soil. Sources include vehicle exhaust, asphalt roads, coal tar, coal, and hazardous waste sites. Most of the health effects of individual PAHs have not been clearly identified. Although they are dissimilar, they tend to appear in groups rather than individually, depending on the process; thus, they are considered a group. The PAH group is also known as Coal Tar Pitch Volatiles or the fused polycyclic hydrocarbons that volatilize from the distillation residues of coal, petroleum, etc. It consists of the following compounds:

- anthracene
- benz(a)anthracene
- benzo(a)pyrene
- benzo(b)fluoranthene
- benzo(g,h,i)perylene

- benzo(k)fluoranthene
- chrysene
- dibenz(a,h)anthracene
- fluoranthene
- fluorene
- indeno(1,2,3-cd)pyrene
- phenanthrene
- pyrene.

The PAH's standards are set for coal-tar pitch volatiles determined as the cyclohexane-extractable fraction. NIOSH's TWA is 100 mg/m^3; OSHA's TWA and ACGIH's TLV are 200 mg/m^3.

Background levels of the PAH group in air in the United States are reported to be in the range of 20–1200 $\mu g/m^3$ in rural areas and 150–19,300 $\mu g/m^3$ in urban areas. Personal air concentrations of benzo(a)pyrene in Padua, Italy, were estimated in winter and summer; the means were 370 $\mu g/m^3$ and 121 $\mu g/m^3$ respectively (Minoia, 1997). Similar measurements were taken on the street and inside a city park in Copenhagen, Denmark; the results were 4400 $\mu g/m^3$ and 1400 $\mu g/m^3$ respectively (Nielsen, 1996). Background levels of PAHs in drinking water are in the range of 4–24 ng/L.

Humans are exposed to PAHs by choice of lifestyle and culture. For example, a study of commercial suntan oils based on mineral or vegetable oils that were analyzed for PAHs showed that all the samples contained benzo[a]pyrene together with other mutagenic, co-carcinogenic or noncarcinogenic PAHs, fluoranthene, benzo[k]fluoranthene, and anthracene. The total PAH content of the samples varied from 89–189 ng/g, while benzo[a]pyrene levels were in the 2–5 ng/g range. The results suggest that users of suntan oils may be exposed to low levels of potentially hazardous PAHs (Monarca, 1982).

In another study, the levels of 13 PAHs were determined in smoked fishery products from both modern smoking kilns with external smoke generation and from traditional smoking kilns. The average benzo(a)pyrene (BaP) concentration in all 35 samples from modern smoking kilns was 0.1 $\mu g/kg$ (wet weight). The sum of other PAHs determined in the study (benz(a)anthracene, chrysene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, dibenz(a,h)anthracene and indeno(1,2,3-c,d)pyrene) was about 4.5 $\mu g/kg$ (wet weight). The BaP levels of the 27 smoked fish samples from traditional kilns ranged from 0.2–4.1 $\mu g/kg$, with a mean value of 1.2 μg BaP/kg. The average concentration of the sum of the carcinogenic compounds was 9.0 $\mu g/kg$ (Karl, 1996). Another dietary

study from Italy calculated that the total dietary PAH intake was 3 μg per day per person, and the total intake of carcinogenic PAHs was 1.4 μg per day per person—high compared to the calculated inhalation of 0.370 μg per day in the most-polluted cities (Lodovici, 1995).

Scant information is available on the human health effects of specific PAH compounds. The limited health-effects data available arises from animal studies, where a wide range of effects have been found but only from exposure to extremely high doses of benzo(a)pyrene by ingestion, dermal contact, or prolonged inhalation (Sharma, 1997). The PAHs in these studies include anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene, and indeno(1,2,3-cd)pyrene. There is no conclusive evidence that similar effects could occur in humans. However, the U.S. Department of Health and Human Services has determined that PAHs may reasonably be considered carcinogens.

Recent epidemiological studies report direct evidence of the carcinogenic effects of PAHs in occupationally exposed subjects. Risks of lung and bladder cancer were dose-dependent when PAHs were measured quantitatively against unexposed control groups. These findings suggest that the current threshold limit value of 200 $\mu g/m^3$ of benzene-soluble matter, which indicates PAH exposure, may be too high; after 40 years of exposure, it gives a relative risk of 1.2–1.4 for lung cancer and 2.2 for bladder cancer (Mastrangelo, 1996). Studies indicate that when binary mixtures of some PAHs are administered, the yield of nuclear anomalies in the mouse gastrointestinal tract is less than expected by simple addition and closer to that expected by averaging the activities of the two PAHs comprising the mixture (Reddy, 1991).

PARTICULATE MATTER

Particulate matter (PM) is the generic term for a broad class of chemically and physically diverse substances that exist as discrete particles (droplets or solids) over a wide range of sizes. Particles originate from a variety of anthropogenic stationary and mobile sources as well as from natural sources. Particles may be emitted directly or formed in the atmosphere by transformations of gaseous emissions such as sulfur oxides (SO_x), nitrogen oxides (NO_x), and VOCs. The chemical and physical properties of PM vary greatly with time, region, meteorology, and source category, thus complicating the assessment of health effects.

Epidemiological studies on the health effects of particulate matter suggest significant short- and long-term toxicity at current ambient levels in the United States. So far, these effects seem to be less influenced by particle composition, inorganic versus organic, and nominal size than by gravimetric estimates of exposure. This appears to contradict the premises of conventional air-pollution

toxicology, which is based on chemical-specific toxicity and the critical role of size in particle potency. The biological mechanism underlying the health effects found with PM_{10} in epidemiological studies is not well understood.

Time-series mortality studies suggest that an increase of 50 $\mu g/m^3$ in the average 24-hour exposure to PM_{10} above the NAAQS is associated with an increased relative risk (RR)[3] that ranges between 1.025 and 1.05 in the general population, and an even higher RR in at-risk subpopulations, i.e., the elderly and those with pre-existing respiratory conditions (Pope, 1996; Schwartz, 1996a, 1996b). The range of PM_{10}-mortality RR across studies may reflect the likely differences in PM_{10} composition as well as differences in PM_{10}-averaging periods considered in the analyses. Recent analyses of the Harvard Six City Study that was conducted in six eastern U.S. cities found larger associations between excess mortality and fine particles ($PM_{2.5}$), than with coarse particles (PM_{10}–$PM_{2.5}$) alone (Schwartz et al., 1996). Moreover, the correlation of excess mortality with coarse-mass particles becomes not significant, except in Steubenville, OH, where the coarse particles are probably predominantly from industrial combustion sources (RR = 1.053 per 25 $\mu g/m^3$ in $PM_{2.5}$).

Many studies have investigated the relationship between hospital admissions, outpatient visits, and emergency room visits for respiratory and heart diseases and PM_{10} in conjunction with other pollutants, e.g., O_3, SO_2, CO, NO_2, H+. These studies have used data from many cities in the United States and Canada (Burnett, 1994; Schwartz, 1994a,b,c, 1995a, 1996a; Gordian, 1996). Chronic obstructive pulmonary disease (COPD), pneumonia, and nonspecific respiratory-disease hospitalizations show moderate but statistically significant RR in the range of 1.06–1.25 when there is an increase of 50 $\mu g/m^3$ in PM_{10} or its equivalent. Although a substantial number of hospitalizations for respiratory-related illness occur in those older than 65, there are numerous hospitalizations for those under 65 as well.

Many of these studies also examine the effect of O_3, and collectively they indicate that ambient O_3 has a significant effect on hospital admission for respiratory causes, with RR ranging from 1.1 to 1.36 per 100 ppb. For a two-pollutant model ($PM_{10} + O_3$), the RR range is 1.04 to 1.54 per 100 $\mu g/m^3$, while individually the RR for O_3 is slightly lower than for PM_{10} (Schwartz, 1995a). The PM_{10} and O_3 effects appear to be independent of each other, with no reduction in the RR for one pollutant after control for the other. Also, there is a suggestion of an effect between PM_{10} and heart disease; there is none for O_3.

[3]Relative Risk (RR) is the ratio of probabilities that a disease will occur among those exposed to a factor to that of those not exposed.

Studies of acute respiratory illness include upper respiratory, lower respiratory, or cough in children and in adults. Studies of upper-respiratory illness do not show consistent results. Three studies show a RR of 1.2 (Pope, 1991, 1992; Hoek, 1993), and another study estimates it as 1.55 (Braun-Fahrländer, 1992). These inconsistencies could be attributed to the difference in populations.

Studies of lower-respiratory disease give RR ranging from 1.10 to 1.28 (Pope, 1991, 1992; Hoek, 1993; Schwartz, 1991a) and another study set it at 2.0 (Schwartz, 1994). Studies of cough were more consistent, with RR ranging from 0.98 to 1.51 (Pope, 1992; Hoek, 1993; Schwartz, 1994; Dusseldorp, 1994). These estimated RR have a larger scatter than the corresponding RR derived from the hospitalization data. This variability is probably due to the former studies' populations including several different subgroups, whereas the hospitalization studies tended to include more uniform populations.

Limited evidence suggests pulmonary function decrements are associated with chronic exposure to particulate matter indexed by various measures—Total Suspended Particulate (TSP), PM_{10}, sulfates, etc. (Spektor, 1991; Ackerman-Liebrich, 1997; Raizenne, 1996). These cross-sectional studies require a very large sample size to detect differences because of the inherent person-to-person variability; therefore, lack of statistical significance cannot be construed as proof of null effect.

Ultrafine Particles

The ultrafine, or nucleation mode, particles have a median diameter of about 0.02 μm. They have an approximately six-order-of-magnitude higher number concentration and a hundred times higher surface-to-volume ratio than a 2.5 μm-diameter particle when inhaled at the same mass concentration. This means, for example, that for the same mass of particles the ultrafine particles will present a much larger surface where gases could adhere and be carried into the lungs via inhalation, potentially increasing toxicity because of the deeper penetration into the lung. Single ultrafine particles occur in the urban atmosphere in high number concentrations, $5x10^4$–$3x10^5$ particles/cm^3, but in low mass concentrations (Brand, 1991). Fortunately, single ultrafine particles are not very stable and eventually aggregate into larger particles, but new particles are always being produced from anthropogenic sources, e.g., gas-to-particle conversion, combustion processes, incinerator emissions, etc.

Although virtually no human studies exist on the health effects of ultrafine particles, some relevant issues have been examined. A recent study on the effect of ambient-particle size on lung function and symptoms in asthmatics suggests that the effects of the number of ultrafine particles (diameter < 0.1 μm) may be

greater than those of the mass of fine particles (diameters of 0.1 to 2.5 μm) (Peters, 1997). An animal study that investigated the surface coating of ultrafine particles suggests that acute exposure to near-ambient concentrations of sulfuric acid under conditions that promote the formation of acid as a surface coating on respirable particles can induce an enhanced nonspecific airway hyperresponsiveness (Chen, 1992).

ACIDIC AEROSOLS

Several major pollution episodes have occurred in this century. Among these are the Meuse Valley, Belgium, in December 1930; in Donora, PA, in October 1948; and in London in December 1952 and December 1962. These specific historical episodes highlighted the major health effects of acidic aerosols on general populations. Unfortunately, no actual ambient air measurements during the first two episodes are available. In the Meuse Valley incident, although many pollutants existed in the atmosphere, the observed health effects were most strongly associated with sulfuric acid (Firket, 1936). More than 60 people died from this acidic fog, over ten times the normal rate, and hundreds suffered respiratory problems. Those especially affected were the elderly, asthmatics, and heart patients.

About 42 percent of the Donora population experienced deleterious effects from its three-day smog episode. Mild upper-respiratory tract symptoms were evenly distributed through all age groups and on average lasted for four days. More than half of those above 55 complained of dyspnea, the most common symptom. The observed health effects could have been produced by two or more contaminants, i.e., SO_2 and its transformation products in combination with other PM components (Schrenk, 1949; Hemeon, 1955).

The London smog episode of 1952 resulted in an estimated 4000 excess deaths. Hospital admissions increased dramatically, mainly among the elderly and those with preexisting cardiac or respiratory disease. Otherwise healthy pedestrians, their vision limited to as little as three feet, covered their noses and mouths in an attempt to minimize their exposure to "choking air" (United Kingdom Ministry of Health, 1954). As a consequence of the 1952 London episode, daily measurements of British Smoke (BS), related to PM and SO_2, started in 1954. These historical pollution data indicate that extremely elevated daily acidic aerosol concentrations (approximately 400 $\mu g/m^3$) may be associated with excess human mortality when present as copollutants with elevated concentrations of PM and SO_2. At non-episode pollution levels ($H_2SO_4 \leq 30$ $\mu g/m^3$), associations between acidic aerosols and mortality in London are statistically significant even though these associations cannot be separated from BS or SO_2. Increased hospital admissions for respiratory causes were also re-

ported (Ito, 1993). Studies in the northeastern United States and Canada, where high levels of acidic aerosols are present during the summer, indicate that the increase in respiratory hospital admissions associated with acidic PM_{10} is about six times that for nonacidic PM_{10} (Thurston, 1994).

Short-term exposures to sulfuric acid (H_2SO_4) aerosols (0.5 μm in diameter) at ambient levels can alter mucociliary clearance, the primary lung defense mechanism. In healthy and asthmatic adults, mucociliary clearance is initially increased at 100 $\mu g/m^3$ of H_2SO_4 and then decreased for higher concentrations (300–1,000 $\mu g/m^3$) (Leikauf, 1984; Spektor, 1985). Pulmonary function was also decreased in adult asthmatics at those exposures (Spektor, 1985). Animal studies support these findings and also show altered resistance to bacterial infection and altered alveolar macrophage function. Low-level H_2SO_4 (100 $\mu g/m^3$) reacts synergistically with O_3 by exacerbating the O_3 lung-function effects. In these controlled laboratory studies, the H_2SO_4 exposures have been controlled nasal exposures (normal daily human exposure is also by mouth) where a significant acid neutralization by ammonia (NH_3) may occur, thus reducing the deposited lung dose (Schlesinger, 1992a, 1992b).

METALS

There are no reported toxicological studies of acute effects of inhaled metals at or below 0.5 $\mu g/m^3$. Most of the data originates in occupational settings and laboratory animals. These have limitations, such as how to extrapolate from animal models to humans; how to separate effects from the confounders probably present in occupational settings such as toxic gases and other inhalable particulate matter; and how to extrapolate effects from high- to low-exposure levels. Still, a review of the available literature regarding the health effects of trace elements at the lowest concentrations is included here. The mean concentrations of trace elements in the PM_{10} distribution—measured at Camp Thunderock, the site with the highest measured levels in the Gulf War region—still are too low to yield any known health effect. These measurements are shown in Table 3.5. For illustration purposes, the range of values in urban, rural, and remote areas of the United States is included. The Camp Thunderock data, except for nickel, fall in the range of rural areas, while nickel is within the urban range. It should be stressed that the levels in the United States are not high, but that levels in the Persian Gulf were very low.

Arsenic

Most of the available human inhalation data is based on occupational exposures to arsenic trioxide. For exposure concentrations above 1,000 $\mu g/m^3$,

Table 3.5

Concentration of Elements Associated with PM in the Ambient Air (ng/m^3)[a]

	United States			
Elements	Urban	Rural	Remote	Camp Thunderock (August 1991)
Arsenic	2–2,300	1–28	0.007–1.9	4.25
Cadmium	0.2–7,000	0.4–1000	0.003–1.1	4.30
Chromium III	2.2–124	1.1–44	0.005–11.2	44.0
Iron	130–13,800	55–14,530	0.62–4160	8390
Nickel	1–328	0.6–78	0.01–60	136.0[b]
Lead	0.007–64	2–1700	30–96,270	587.0
Vanadium	0.4–1460	2.7–97	0.001–14	38.8
Zinc	15–8328	11–403	0.03–460	107.0

SOURCE: USAEHA, 1994.

[a] $ng/m^3 = 0.001\ \mu g/m^3$.

[b] Mean concentrations in the Gulf region were in the range of rural areas in the U.S. except for nickel, which was at the urban level.

symptoms include severe irritation of the nasal mucosa, larynx, and bronchia (Holmqvist, 1951; Pinto, 1953). These irritations may lead to hoarseness, laryngitis, bronchitis, and sometimes perforation of the nasal septa (Pinto, 1953). Increased peripheral vasospastic and Raynaud's syndrome were found in Swedish arsenic workers (Lagerkvist, 1986).

Beryllium

As the production of missiles, nuclear devices, and electronics grew and modern industrial technologies emerged, the risk of occupational exposure to beryllium became widespread. The environmental burden also increased as a result of emissions from plants producing and processing beryllium or its alloys and compounds. The major exposure to beryllium is through inhalation, which induces specific sensitization and nonspecific effects leading to chronic beryllium disease (CBD). CBD is an immunologically mediated granulomatous and fibrotic pulmonary disorder with increased numbers of lymphocytes in bronchoalveolar lavage fluid similar to that found in hypersensitive pneumoconitis. It has been hypothesized that epithelial injury and permeability changes occur early in CBD and are indicative of disease severity (Inoue, 1997). Associated symptoms are dyspnea on exertion, cough, chest pain, weight loss, and general weakness.

Cadmium

Health effects of cadmium exposure at ambient airborne concentrations have not been reported. The observed health effects in humans and animals are at concentrations three or more orders of magnitude higher than ambient. Above a critical threshold of 1000 $\mu g/m^3$ per year, there is evidence of kidney damage, i.e., proteinuria (Mason, 1988). Acute respiratory effects of inhaled cadmium have been reported as pneumonitis and edema for exposures of approximately 300 $\mu g/m^3$ for extended periods. Several studies found a statistically significant excess risk of lung cancer in the highest exposure groups (Elinder, 1985; Sorahan, 1987; Thun, 1985); thus, the International Agency for Research on Cancer (IARC) classified cadmium as a human carcinogen (IARC, 1993). Similar results were obtained from animal studies confirming that inhalation exposure to cadmium compounds can result in respiratory tract injury. Exposure to very high levels of cadmium has been shown to lead to a decreased immune response in mice (Graham, 1978).

Chromium

Chromium, like many transition metal elements, is essential to life at low concentrations, yet toxic at higher concentrations. In addition to the overt symptoms of acute chromium toxicity, delayed manifestations of chromium exposure become apparent by subsequent increases in the incidence of various human cancers. Studies show conclusively that chromium in its hexavalent form (Cr (VI)) is both toxic and carcinogenic; in its trivalent form (Cr (III)) it is not, and moreover is essential in the metabolism of insulin (Bencko, 1985). Chromium (VI) easily crosses cell membranes and exerts genotoxic effects. The available evidence strongly indicates that Cr (VI) reduction in body fluids and long-lived non-target cells is expected to greatly attenuate its potential toxicity and genotoxicity, to imprint a threshold character to the carcinogenesis process, and to restrict the possible targets of activity.

On the other hand, no metabolic oxidation of Cr (III) has been observed. The Cr (VI) sequestering capacity of whole blood and the reducing capacity of red cells explain why this metal is not a systemic toxicant except at very high doses. Reduction by fluids in the digestive tract, i.e., saliva and gastric juice, and sequestering by intestinal bacteria account for the poor intestinal absorption of Cr (VI). Chromium (VI) escaping reduction will be detoxified in the blood and liver. These processes explain the poor toxicity of Cr (VI) and its lack of carcinogenicity when introduced orally or swallowed following reflux from the respiratory tract. The chemical environment in the gastrointestinal tract and the blood is effective even under fasting conditions in reducing Cr (VI) to one or more forms of Cr (III) (Kerger 1997). Inhaled Cr (VI) is reduced in the epithelial

lining fluid and in the pulmonary alveolar macrophages. The lung parenchyma has reducing capacity with slightly higher specific activity than the bronchial tree. Therefore, even in the respiratory tract, the only consistent target of Cr (VI) carcinogenicity has barriers hampering its carcinogenicity. This protection could be overcome only by massive exposure through inhalation, as in work environments lacking proper industrial hygiene (De Flora, 1997).

Iron

Most of the human data are based on occupational exposures to iron oxide, with effects limited to respiratory symptoms and dysfunction, and no data are available on acute exposures. Mining and smelting processes generate iron oxides, silica, and other substances. Iron oxides deposited in the lung result in changes in lung X-rays. This effect is known by many names: siderosis, iron pneumoconiosis, hematite pneumoconiosis, iron pigmentation of the lung, and arc welder's lung. Siderosis is prevalent in 5–15 percent of iron workers exposed for more than five years (Sentz, 1969) and in a reported 34 percent in workers exposed to ferric oxide dust at concentrations ranging from 3500 to 269,000 $\mu g/m^3$ (Teculescu, 1973). Evidence of lung fibrosis was not observed, but chronic cough was reported by 80 percent of the workers. Several studies reported high incidence of lung cancer mortality, but in all cases there was co-exposure to other potential carcinogens (Boyd, 1970). Animal studies that resulted in respiratory-tract cell injury and alveolar fibrosis were conducted at concentrations of 14,000 $\mu g/m^3$ for one month (Nettesheim, 1975). Iron oxide particles have been the carrier particles for radioactive tracers, i.e., Technetium (^{99m}Tc) and Gold (^{198}Au), in many human and laboratory animal studies designed to measure different aspects of pulmonary-particle deposition and clearance. These exposures were brief, but the concentrations were orders of magnitude higher than ambient levels. There are no reports of acute effects (Leikauf, 1984; Brain, 1991).

Lead

Children show a greater sensitivity to lead's effects than do adults, because children absorb and retain more lead in proportion to their weight. The most sensitive target of lead poisoning is the developing brain. Lead exposure at age two will result in continued deficits in neurologic development, such as lower IQ scores and cognitive deficits, at age five (Needleman, 1990; Schwartz, 1987). The primary sources of environmental exposure to lead are leaded paint, auto emissions, drinking water from plumbing leachate, and ceramic ware. Once in the bloodstream, lead is distributed in the blood, soft tissue, and mineralizing tissue. Bones and teeth of adults contain more than 95 percent of the body's

lead content. Lead affects primarily the peripheral and central nervous system, blood cells, and the metabolism of vitamin D and calcium; it also causes reproductive toxicity (Gerber, 1980). Levels of lead in ambient air range from 7.6 x 10^{-5} $\mu g/m^3$ in remote areas like Antarctica (Maenhaut, 1979) to more than 10 $\mu g/m^3$ near sources such as a smelter, with an average annual concentration of below 1 $\mu g/m^3$ for urban sites. The NAAQS for lead is 1.5 $\mu g/m^3$ (EPA, 1997).

Mercury

Mercury is ubiquitous in the environment, and it is also released in industrial activities and combustion of fossil fuels. Mercury so released is in an inorganic form, predominantly as metallic vapor. In aquatic environments, it is microbiologically transformed into the organic compound methylmercury. Therefore, populations with higher intake of foods originating in water have higher exposure to methylmercury (Hansen, 1997). In the past, methylmercury compounds were manufactured as fungicides or as unwanted byproducts in the chemical industry. Methylmercury absorbed from the diet distributes within a few days to all tissues in the body. It crosses the blood-brain and placenta barriers to reach its main target tissue, the brain. The biological half-life in human tissues is about 50 days, and mercury is eliminated chiefly in the feces after conversion to inorganic forms. Adult poisoning is characterized by focal damage to discrete anatomical areas of the brain such as the visual cortex and granule layer of the cerebellum. A latent period of weeks or months may lapse before signs and symptoms appear. The latter manifestation is paresthesia, ataxia, constriction of the visual fields, and hearing loss. Presently a chief concern is with the more subtle effects arising from prenatal exposure such as delayed development and cognitive changes in children (Clarkson, 1997).

Nickel

In order of abundance in the earth's crust, nickel ranks as the 24th most common element, and it is found in different media in all parts of the biosphere. Nickel is a useful metal, particularly in various alloys, in batteries, in nickel plating, as a catalyst, and in pigments. Occupational exposure may lead to the retention of 100 μg per day. Environmental levels depend on emissions from nickel-manufacturing industries and airborne particles from combustion of fossil fuels. Absorption from ambient pollution is a minor concern. Vegetables, legumes, and nuts contain nickel, and the average dietary intake is 200 to 300 μg per day (Grandjean, 1984). Owing to the low absorption rate, nickel compounds in the gastrointestinal tract (except for nickel carbonyl) are essentially nontoxic after ingestion.

Some nickel compounds have been found to be carcinogens. Nickel carbonyl is correlated to nasal and lung cancer. Nickel subsulfide may be the most potent nickel carcinogen, but exposures to this compound are limited to specific occupations (Norseth, 1980). Other clinical manifestations include acute pneumonitis from inhaled nickel carbonyl, chronic rhinitis and sinusitis from inhaled nickel aerosols, and dermatitis and other hypersensitivity reactions from dermal exposures to nickel alloys (Sunderman, 1977). Cutaneous nickel allergy (contact dermatitis) affects 15–20 percent of the female general population and 1 percent of the males (Savolainen, 1996).

Vanadium

Most of the reported exposures are to vanadium pentoxide dusts in occupational settings. Worker exposure to vanadium dusts ranging in duration from several hours to several years, in concentrations from 100 to 300 $\mu g/m^3$, produced temporary mild respiratory distress, i.e., productive cough, wheezing, chest pain, runny nose, or sore throat (Lewis, 1959). At concentrations ranging from 1,000 to 6,500 $\mu g/m^3$ for 1–2 years, lower respiratory tract effects were observed, such as rhinitis, nasal discharge, irritated throat, bronchopneumonia, and asthmatic bronchitis (Sjoberg, 1950; Levy, 1984). Relatively low acute exposures—60 $\mu g/m^3$ and 100 $\mu g/m^3$ for 8 hours—produced cough and mucus formation that lasted for one week (Zenz, 1967). Acute and chronic laboratory animal studies show that the respiratory tract is the main target of inhaled vanadium compounds (Lee, 1986; Knecht, 1985).

Zinc

Inhalation of zinc (mostly from zinc oxide fumes) may produce significant pulmonary irritation and inflammation, also known as metal fume fever. Since zinc has low toxicity, the exposure concentrations have to be in the mg/m^3 range to induce these symptoms. Fever, chills, chest tightness, muscle/joint pain, sore throat, headache, and increased airway resistance were reported 4 to 8 hours after exposure to 4900 $\mu g/m^3$ for 2 hours. Multiple higher-exposure concentrations resulted in the development of adaptation or tolerance after the initial symptoms of zinc fume fever subsided (Gordon, 1992).

PHOTOCHEMICAL POLLUTION

Photochemical smog, or pollution, arises from a series of complex atmospheric reactions that result in a mixture of ozone, nitrogen oxides, aldehydes, peroxyacethyl nitrates, and reactive hydrocarbons. If sulfur dioxide is present, sulfuric acid droplets may be formed, as nitric-acid vapor can be formed from NO_2.

Hydrocarbons, under normal circumstances, are of much less concern because their concentrations are too small to become toxic. However, they are important because of the role they play in the formation of photochemical smog. In simple terms, ultraviolet light (UV) splits molecular oxygen, O_2, into atomic oxygen, O, to combine with other O_2 molecules to form O_3. At the same time, in the troposphere, NO_2 absorbs UV to form O and NO. This O combines with O_2 to form O_3 and O_3 with NO forms NO_2. This process is cyclic. In the absence of hydrocarbons, this series of reactions would reach a steady state with no excess of O_3. The hydrocarbons are attacked by the free atomic O resulting in oxidized compounds and free radicals that react with NO to produce more NO_2 and therefore a build-up of O_3. This process occurs when the automobile emissions of morning commuters interact with the sun's UV to produce smog by noon.

Ozone

Ozone (O_3) is found in the free troposphere and in the planetary boundary layer (PBL), the layer next to the surface of the earth. Background O_3 in the PBL occurs as the result of incursions from the stratosphere and through photochemical formation from precursors, CO, VOCs, and NO_x. These precursors are all associated with combustion processes.

Short-term O_3 exposure in healthy humans induces changes in pulmonary function, decreased volumes and flows, increased airway responsiveness, and airway irritation such as cough or pain on deep inspiration. Asthmatics experience similar effects and increased wheezing (Linn, 1994; Koenig, 1987, 1988; Molfino, 1991; Kulle, 1984). Inflammatory responses have been observed after acute exposures to O_3 at concentrations found in U.S. cities (Devlin, 1990, 1991, 1996; Koren, 1990, 1991). Recovery from acute exposure is usually complete within 24 hours of the end of exposure. There is evidence of a plateau in lung volume in response to prolonged O_3 exposure. Also, available data indicate that exposure to O_3 for months and years causes structural changes in several regions of the respiratory tract. Research to date indicates that the area most affected is the centriacinar region, where alveoli and conducting airways meet (Sherwin, 1991).

There are very few human studies on binary pollutant exposure. Ozone in combination with SO_2, H_2SO_4, HNO_3, NO_2, or peroxyacetyl nitrate (PAN) causes an additive response on lung spirometry or symptoms (Dreshsler-Parks, 1989; Aris, 1991; Hazuka, 1994; Utell, 1994; Koenig, 1990, 1994). Animal studies of O_3 and NO_2 or H_2SO_4 show that effects can be additive, synergistic, or even antagonistic, depending on the endpoint studies (Gelzleichter, 1992; Warren, 1986; Schlesinger, 1992a, 1992b). The chronic effects of co-pollutant exposure are still not understood. There is evidence suggesting that people with preexisting

limitations in pulmonary function and exercise activity, e.g., asthma, COPD, chronic bronchitis, and ischemic heart disease, are at risk from O_3 exposure.

Nitrogen Dioxide

Nitrogen dioxide (NO_2), like O_3, is a deep-lung irritant, but less potent. No alterations in lung function were observed in healthy humans after exposure to NO_2 for up to 4 ppm for three hours; at 1.5–2 ppm, however, slightly enhanced airway reactivity was shown (Mohsenin, 1987). It is interesting to note that ascorbic-acid pretreatment protected the subjects from this hyperreactivity (Kjaergaard, 1996). Animal studies have shown associations between NO_2 and both viral and bacterial infections, suppression of macrophage, and alterations in lung clearance (Schlesinger, 1987). NO_2 is also an important indoor air pollutant, especially in homes with unvented gas stoves or kerosene heaters.

Sulfur Dioxide

Sulfur dioxide (SO_2) is a gas formed when fuels containing sulfur, mainly coal and oil, are burned, and also during smelting and other industrial processes. SO_2 readily oxidizes to sulfate in the atmosphere in the presence of catalysts—e.g., metals such as iron, manganese, and vanadium in dispersing smokestack plumes, or via photochemical processes. However, most of the oxidation of SO_2 occurs in the atmosphere, where it is transformed into sulfuric acid (H_2SO_4). As such, it may undergo long-range transport to areas hundreds of miles away from the emission source. SO_2 is an upper-airway irritant that can stimulate bronchoconstriction and mucus secretion. Animal studies indicate that relatively low concentration exposures of SO_2 (0.1 to 20 ppm) for long periods have marked effects consistent with bronchitis (Nadel, 1965).

Carbon Monoxide

Carbon monoxide (CO) comes from both natural processes (approximately 40 percent, mostly through oxidation of hydrocarbons but also from plants and oceans) and from anthropogenic processes such as combustion of fossil fuels and oxidation of methane (approximately 60 percent). CO, an asphyxiant, is readily absorbed through the lungs into the blood stream, where it competes with O_2 to bind to hemoglobin (Hb) in red blood cells, forming carboxyhemoglobin (COHb). Its toxicity arises from its high affinity to Hb, approximately 240 times greater than that of O_2 (Haldane, 1898). A unique characteristic of CO exposure is that the blood COHb level is a very useful biomarker of exposure. The level of COHb may be determined directly by blood analysis and also estimated by measuring the CO concentration in exhaled breath. COHb levels can

be calculated from known CO exposures by solving the nonlinear differential equation known as the Coburn equation (Coburn, 1965).

There is a baseline blood level of COHb of approximately 0.5 percent for healthy adults and 1–8 percent for smokers. The most sensitive members of the general population to CO exposure are those with ischemic heart disease. They will start experiencing reduced exercise duration due to increased chest pain (angina) at CO levels that will give them 3–6 percent COHb (Kleinman, 1989; Allred, 1991). Healthy individuals will experience a reduction in their maximal exercise performance at CO levels that, after one hour, give them COHb levels of 2–3 percent. At COHb blood levels greater than 5 percent, healthy individuals may have equivocal effects on visual perception, audition, motor and sensory performance, vigilance, and other measures of neurobehavioral performance. At higher blood COHb levels, 10 percent or more, neurological effects could occur, including headache, dizziness, weakness, nausea, and confusion. Unconsciousness and death can occur with continuous exposure to high CO levels in a workplace or in unventilated rooms with faulty unvented combustion appliances.

Hydrogen Sulfide

The acute effects of exposure to H_2S are well recognized. Odor of "rotten eggs," followed by olfactory paralysis, mucosal irritation, and keratoconjunctivitis are the typical effects of H_2S at lower concentrations. H_2S-induced acute central toxicity leads to reversible unconsciousness called a "knockdown." It has been suggested that repeated or prolonged knockdowns are associated with chronic neurological sequelae (Guidotti, 1994). Knockdowns can be fatal due to respiratory paralysis, cellular anoxia, and pulmonary edema. There are some indications that other chronic health effects include neurotoxicity, cardiac arrhythmia, and chronic eye irritation, but apparently not cancer. Concentrations above 50 ppm—five times the occupational limit—can cause death (Kilburn, 1995). Healthy human volunteers inhaled air with 10 ppm H_2S in a blind test in-between 30-minute exercise sessions at 50 percent of their maximal oxygen. All experienced a significant decrease in oxygen uptake with concomitant increase in blood lactate. However, no significant changes were observed in arterial blood parameters (Bhambhani, 1997), pulmonary function, or diffusion capacity (Bhambhani, 1996).

LONG-TERM EXPOSURE TO AIR POLLUTION (CANCER)

The role of inhaled pollution in human lung cancer is difficult to assess because the vast majority of respiratory cancers result from cigarette smoking. Volatile organic compounds and nitrogen-containing and halogenated organic com-

pounds account for most of the compounds that have been studied with animal and genetic bioassays. Most of these compounds are derived from combustion sources, from power plants to incinerator emissions. Other potential carcinogens also result from mobile sources as products of incomplete combustion and their atmospheric transformation products, as well as fugitive or accidental chemical releases.

The carcinogen potency of air pollution resides usually in the particulate fraction. Polycyclic organic chemicals and semivolatiles are associated with the particulate fraction and could have a prolonged residence time at sensitive sites in the respiratory tract when inhaled. Genetic bioassays have demonstrated potent mutagenicity, and presumably carcinogenic potential, of various chemical fractions of ambient aerosols. Copollutants such as irritant gases that initiate inflammation may promote carcinogenic activity (Lewtas, 1993).

Populations at Risk

Groups within the general population most clearly at risk include the elderly and those with cardiopulmonary diseases. Epidemiological studies indicate that mortality and morbidity from respiratory causes are strongly related to ambient PM exposures. Possible mechanisms include airway narrowing, increased mucous secretion or increased viscosity, and inflammation and epithelial cell damage in persons with respiratory disease. Cardiac arrhythmia has been hypothetically linked to mortality due to acute PM exposure. Risk of mortality due to lower-respiratory disease, e.g., pneumonia, is increased by ambient PM exposure by both exacerbation and increasing susceptibility to infectious disease by decreasing clearance, impairing macrophage function, or other specific or nonspecific effects on the immune system. Smokers comprise about 80 percent of individuals with COPD, and together with a small but notable portion of patients with cardiovascular disease, are likely to be at increased risk for PM health effects. Asthmatics are more responsive than nonasthmatics to acidic aerosols; asthma exacerbation, even requiring medical care, is associated with PM_{10} exposure.

HEALTH EFFECTS STUDIES CONDUCTED DURING THE OIL FIRES

Only a few quantitative studies were performed during the Persian Gulf War to assess the possible health effects of the oil well fires. Four are described here. One compared U.S. personnel in Kuwait City, oil firefighters, and a control group. Another analyzed the genotoxicity of the soot. A third study examined feral cats in Kuwait City and Ahmadi. The fourth was comprised of several self-administered health questionnaires. The USAEHA also estimated cancer and noncancer risks due to the environmental exposure levels it measured using an

EPA methodology[4], finding the risks similar to those for the general U.S. population (USAEHA, 1994).

Firefighters

This study documented VOC levels in the blood of U.S. personnel in Kuwait City in May 1991 (Group I) and American firefighters working in the oil fields in October 1991 (Group II). Concentrations of VOCs in both groups were compared with a random sample of persons in the United States (Control Group). The median concentration of VOCs in Group I were equal to or lower than those in the Control Group. However, significant differences were found with Group II. Median levels of ethyl-benzene were 10 times higher than in the Control Group, while benzene, toluene, and xylene levels were more than double those of the Control Group. These VOC levels are comparable to those measured in German workers in Kuwait. The results were drawn from a small sample of volunteers. Considering that the half-life of these compounds in blood is less than 4 hours, they may be taken only as a suggestion of what the VOC-exposure levels were in Kuwait during the fires (Etzell, 1994). This work is summarized in Table 3.6.

Genotoxicity

The in-vitro genotoxicity of soot from the Kuwait oil fires was also studied. Dose-dependent increases were observed for both sister chromatoid exchanges in human peripheral blood lymphocytes and mutation of the hprt locus in the

Table 3.6

VOC Concentrations in Blood in U.S. Personnel (μg/l)

VOC	Kuwait City Personnel (Group I)	Firefighters (Group II)	U.S. Reference (Control)
Benzene	0.035	0.18	0.066
Ethyl-benzene	0.075	0.53	0.052
m,p-Xylene	0.14	0.41	0.18
o-Xylene	0.096	0.26	0.10
Toluene	0.24	1.5	0.30

SOURCE: Etzell, 1994.

[4]As part of the Superfund Program, the EPA developed reference dose values (RfCs) for many toxic agents that estimate a daily exposure level for the general population that is likely to be without appreciable risk during a specific period. These estimates have been used to calculate cancer and noncancer risks (EPA, 1992).

metabolically competent human lymphoblast cell line AHH-1. Similar magnitudes were observed when testing air particulate isolated from Washington, DC. Using ^{32}p-postlabeling assay, no increase in DNA adduct formation was observed in AHH-1 cells treated with particulates isolated from sampling in Kuwait (Kelsey, 1994).

Feral Cats

A study to assess histopathological lesions and analyze chemical accumulations from exposure to the smoke from the Kuwait oil fires collected 12 adult feral cats from Kuwait City and 14 from Ahmadi. Specimens from the lungs, liver, and kidneys, as well as urine and blood samples were examined. The pharyngeal mucosa in all animals was normal. Neither hyperplasia of the epithelium nor cellular atypia were observed. Minimal changes were observed in the larynx. Only two of the 26 cats examined had hyperplasia of the submucosal glands and both were from Kuwait City. No major organs displayed lesions that could be attributed to breathing smoke from the oil well fires or to hydrocarbon inhalation.

Although the results from the heavy-metal analysis were difficult to interpret, because of a lack of "normal" ranges for cats as well as ignorance about what is "normal" for the region, the results seem reasonable when compared to other animal species. Analyses for vanadium and nickel were within normal limits, suggesting that the smoke was not a serious health risk. The clearance mechanism of the ciliated region of the lung apparently was efficient at eliminating particulate matter from the smoke. None of the lesions associated with prolonged exposure to respiratory irritants, and consistent with chronic bronchitis, chronic obstructive pulmonary disease, pulmonary fibrosis, or emphysema, was observed (Moeller, 1994).

Health Survey Questionnaires

A self-administered symptoms and medical-history questionnaire was given to the 11th Armored Cavalry Regiment, then based in Fulda, Germany. A group of 331 soldiers (Group I) completed a survey prior to deployment during the period beginning just before departure from Germany and ending about eight weeks after arrival in Kuwait, a total of 12 weeks. Another 1599 soldiers (Group II) completed a postreturn survey about four weeks after returning to Germany, which included questions on symptoms before, during, and after the mission. The symptoms that appeared once or a few times during the first eight weeks in Kuwait but were not experienced before going to Kuwait were headache (55 percent), lightheadedness (48 percent), fatigue or weakness (45 percent), skin rashes (41 percent), and diarrhea (42 percent). About 35 percent of those who

did not usually have cough or phlegm first thing in the morning before deployment reported the symptom after arriving in Kuwait.

The respondents in Group II recalled their complaints in Kuwait and compared them to those following their return. They reported that the persistence of symptoms was higher in Kuwait than during the eight weeks following their return to Germany. The increase in complaints (percent in Kuwait minus percent in Germany) for occasional symptoms were: eye irritation (27 percent), burning eyes (25 percent), shortness of breath (17 percent), weakness or fatigue (15 percent), skin rashes (14 percent), and respiratory irritation (14 percent). Symptoms were related to reported proximity to the oil fires, and their incidence generally decreased after the soldiers left Kuwait (Petruccelli, 1997).

Another self-administered symptoms questionnaire was developed by the U.S. Navy, and was completed by 2668 servicemen during March 28–31, 1991. Females were excluded because of their very small number. The respondents were divided into three categories according to their proximity to the oil well fires and duration of exposure. Group I consisted of 892 Marines who had the longest exposure, about five weeks at the time of the survey, and were closest to the burning oil wells. Group II included 978 Marines who had a short exposure to the oil fires, and were then stationed in Saudi Arabia about 120 km south of the Kuwait border. Depending on wind conditions, smoke from the oil fires would still have been clearly visible. Group III consisted of 831 Marines who had no direct exposure to the oil fires, having been located in southern Kuwait about 200 km south of the nearest oil field, where only a distant haze was visible on the horizon.

Marines in Group I reported the highest rate of gastrointestinal episodes and respiratory symptoms, followed by Group II. Similar patterns were observed for burning and red eyes. Adjusting for flu vaccination, history of respiratory disease, and smoking status, Group I reported wheezing, coughing, runny nose, and sore throat significantly more frequently than Group III. Group II reported significantly fewer colds than Group III. Smokers reported more complaints than nonsmokers. No patterns were observed when prevalence of respiratory symptoms within groups were examined by job class. The prevalence of wheezing, coughing, and runny nose for each group decreased from Group I to Group II to Group III, the latter being away from the smoke and on the coast away from blowing sand and dust (DoD, 1993).

A study was undertaken to assess and monitor the effects of the oil well fires on a squadron of 125 British bomb-disposal engineers who remained in Kuwait for five months. All subjects completed a health questionnaire and had their respiratory function measured. From June to October 1991, measurements were taken every two weeks; the subjects repeated the questionnaire just before re-

turning to Britain. When data were stratified according to either smoking history prior to deployment or by amount smoked during the tour, no significant differences in either lung-function indices or symptoms were detected when compared to predeployment values (Coombe and Drysdale, 1993).

STUDIES CONDUCTED AFTER THE GULF WAR

CCEP Study

The Department of Defense Comprehensive Clinical Evaluation Program (CCEP) provides medical evaluation to Gulf War veterans now on active duty or retired, members of the reserve components who are Gulf War veterans, and eligible members of families who are experiencing illnesses that they allege to

Table 3.7

Frequency Distribution of Diagnoses (percent)

Diagnostic Categories (ICD-9-CM Code)	Primary Diagnosis: Males[c]	Primary Diagnosis: Females[d]	Primary Diagnosis: All[e]	Any Diagnosis: All[e]
Psychological conditions (230–219)	18.3	19.1	18.4	36.0
Symptoms, signs, and ill-defined conditions (780–799)[a]	18.1	16.5	17.9	43.1
Musculoskeletal system disease (710–739)	18.6	15.9	18.3	47.2
Healthy[b] (V65.5)	9.9	8.6	9.7	10.2
Respiratory system diseases (460–519)	6.9	6.1	6.8	17.5
Digestive system diseases (520–579)	6.5	4.9	6.3	20.4
Skin and subcutaneous tissue diseases (680–709)	6.3	6.0	6.2	19.9
Nervous system diseases (320–389)	5.3	8.8	5.7	17.8

[a]Includes conditions categorized according to ICD-9-CM nomenclature of cases for which no diagnosis was classifiable elsewhere; no more specific diagnosis could be made; signs or symptoms proved to be transient; cases in which a precise diagnosis was not available. [b]Includes registered participants without complaint or sickness as well as those diagnosed as normal or healthy. [c]N=15,944. [d]N=2131. [e]N=18,075.

be related to their service in the Persian Gulf. As of December 1995, more than 27,000 individuals had enrolled in the program, and as of April 1996 18,598 had completed the evaluation process and had their health records entered into the CCEP database. CCEP participants report a wide variety of symptoms spanning multiple-organ systems in no consistent, clinically apparent pattern. The symptoms being reported in the CCEP are not unique; fatigue, joint pain, headache, or sleep disturbances are common among CCEP participants. The frequency distribution by category of diagnosis assigned by the CCEP is presented in Table 3.7.

The order of these diagnoses was determined by usual clinical practice, basing the ranking on the most severe conditions relative to the patient's chief complaints. The most prevalent primary diagnostic categories, accounting for 67.7 percent of the participants, were psychological conditions (18.4 percent); musculoskeletal and connective tissue diseases (18.3 percent); symptoms, signs, and ill-defined conditions (17.9 percent); respiratory diseases (6.8 percent); and digestive system diseases (6.3 percent). Nearly 10 percent received a diagnosis of healthy. When both primary and secondary diagnoses were considered, similar patterns emerged. The most common categories were musculoskeletal diseases (47.2 percent); symptoms, signs, and ill-defined conditions (43.1 percent); psychological conditions (36.0 percent); digestive system diseases (20.4 percent); skin and subcutaneous diseases (19.9 percent); respiratory diseases (17.5 percent); and nervous system diseases (17.8 percent).

Up to seven diagnoses, including healthy, could be reported (one primary and up to six secondary). Among the participants, 19.9 percent had only 1 diagnosis, 18.7 percent had 2, the median was 3, the mean was 3.4, and 9.1 percent were given 7 diagnoses.

Birth Defects

A recently published study on the risk of birth defects among children of Persian Gulf War veterans analyzed the live-birth records at 135 military hospitals over three years (1991–1993). In this period, 33,998 infants were born to Gulf War veterans and 41,463 to nondeployed veterans in military hospitals. The overall risk for birth defects was 7.45 percent, and the risk for severe birth defects was 1.85 percent. There were no significant differences between children born to deployed and nondeployed veterans. When the same definitions of birth defects were applied to the approximately 320,000 live births that occurred nationwide in 1992, the risk of any birth defect was 8.4 percent and the risk of a severe defect was 1.9 percent. The birth defect rates for veterans parallel those of the general population. There was no significant association for ei-

ther men or women between service in the Gulf War and risk of any birth defect (Cowan et al., 1997).

Iowa Study

Several studies have been conducted to assess prevalence of self-reported symptoms, illnesses, and psychiatric conditions among military personnel deployed in the Persian Gulf. The Iowa Persian Gulf Study surveyed 4886 subjects belonging to one of the following groups: Gulf War regular military, Gulf War National Guard/Reserve, non–Gulf War regular military, and non–Gulf War National Guard/Reserve. The results indicate that, compared to non–Gulf War military, Gulf War veterans reported a significantly higher presence of symptoms of depression, post-traumatic stress disorder (PTSD), chronic fatigue, cognitive dysfunction, bronchitis, asthma, fibromyalgia, alcohol abuse, anxiety, sexual discomfort, and diminished mental and physical functional health. Within the National Guard/Reserve, the differences in prevalence for the above-listed symptoms between Gulf War veterans and non–Gulf War veterans are higher than for comparable groups in the regular military. For asthma and bronchitis there is a statistically significant (2.3 percent) difference between Gulf War military and non–Gulf War military (Iowa Persian Gulf Study Group, 1997).

The Iowa Persian Gulf Study Group is performing a follow-up study that includes a complete medical evaluation of a small random sample of the original cohort to verify the sensitivity and specificity of the larger study. The medical evaluation includes pulmonary-function tests that will also aid in verifying the small difference in lung-disease prevalence between the two military groups (Iowa Persian Gulf Study Group, 1997).

One must look cautiously at these studies because none includes quantitative measures of exposure or effects; findings are based solely on self-reported survey questionnaires administered years after the war's end. Some survey studies conducted years later probably have large uncertainties due to different recall and other biases.

CONCLUSIONS

The military personnel who were deployed in the Gulf were healthy and, for the most part, young. Thus, one could argue that the preceding discussion on the possible health effects due to exposures to pollutants present in the Gulf would not apply to Gulf War veterans to the same extent they would to susceptible populations. Moreover, these veterans' exposure levels, except for PM_{10}, were much lower than the U.S. occupational standards, and even lower than ambient

air standards. The exposure concentrations of the pollutants measured in the Gulf, except for PM_{10}, were lower than those in U.S. urban areas. Thus, one would not expect this population to experience ill health effects as a result of exposures in the Gulf.

Several factors may have made Gulf War veterans more vulnerable to pollutants. A significant number of Gulf War veterans were smokers and cannot be considered as having no lung impairment. Smokers react differently than nonsmokers to inhaled pollutants, even at a low concentration of irritants. Also, a small percentage of the veterans may have had some underlying predisposition to pulmonary disease such as asthma that might be triggered after exposure to high levels of PM_{10}.

The levels of PM_{10} were extremely high, not because of emissions from the oil fires, but from the desert sand. These high levels of PM_{10} may perhaps explain the preliminary findings of the Iowa Study regarding respiratory symptoms as well as some of the respiratory symptoms reported in the CCEP. Although there are personal communications indicating that there were increased respiratory complaints among the indigenous population during the oil fires, no evidence of health effects or epidemiological studies were found in the peer-reviewed literature.

Chapter Four

FINDINGS AND CONCLUSIONS

The concentrations of VOCs, PAHs, metals, and criteria pollutants in the Gulf region were much lower than initially presumed, considering the magnitude of the fires. The measurements of the mean concentrations of these pollutants are consistent across studies. The maximum concentrations due to oil well fire emissions measured in the Gulf region are comparable to levels found in suburban locations in the U.S., lower than those found in large urban centers in the U.S., and much lower than the U.S.-recommended occupational levels. Based on the present review of what is known from animal and human exposure and epidemiological data, the concentrations of VOCs, PAHs, metals, and criteria pollutants found in the Gulf region during the Kuwait oil well fires were much lower than the levels that are known to cause short- or long-term health effects. Even assuming a worst-case scenario—that all troops were exposed to the measured concentrations of the contaminants of interest for six months, all was absorbed, nothing was eliminated—the cumulative dose of VOCs, PAHs, and other pollutants would still fall below the levels known to cause any of the health effects described in previous sections.

The health surveys conducted during the oil fires indicate increased symptoms and an association between prevalence of complaints and proximity to the oil fires. As yet, there are no data to support those symptoms being indicators of disease or that the symptoms were associated with proximity to the oil fires.

Particulate matter, however, was found to be extremely high at all monitoring sites compared with values in the U.S. The Gulf region is part of the Arabian desert, characterized by high concentrations of very fine sand dust as well as strong winds—particularly during the season of the Shammal winds (from March to September). Because troops are still stationed in the Gulf region, CHPPM continues to monitor ambient levels of contaminants, thus recording environmental concentrations for the region. These data allow for separating exposures due to emissions from the oil well fires from environmental exposures ubiquitous to the region.

Figure 4.1 illustrates the mean monthly PM_{10} concentrations (black bars) from limited measurements during 1994 in Kuwait City (Kirkpatrick, 1997). For comparison, Figure 4.1 also includes the monthly PM_{10} concentrations measured during the Gulf War in Camp Thunderock by the (then) USAEHA (USAEHA Interim report). The black bars clearly show the high-PM_{10} mean concentrations, as well as the arrival of the Shammal winds in March and their gradual disappearance during September–October. The gray bars show that, even in June 1991, when 500 oil wells were still burning, the mean monthly PM_{10} concentrations were not dramatically higher than is common for that time of the year, as they were originally predicted. However, because these data represent monthly means from limited one-year measurements, they can only be considered as indicators of trends.

Daily PM_{10} concentrations varied dramatically, from less than 100 $\mu g/m^3$ on one day to 1200 $\mu g/m^3$ the next, as illustrated in Figure 4.2. The chart also shows the rate at which the oil fires were extinguished (triangles).

A large concentration of dust in itself may be inconsequential as a health risk unless the dust contains a significant amount of PM_{10} particles. Inhaled particles of 2.5–10 μm deposit in the ciliated portion of the lung and are removed by the "mucociliary escalator" in about 24 hours. Particles larger than 10 μm are

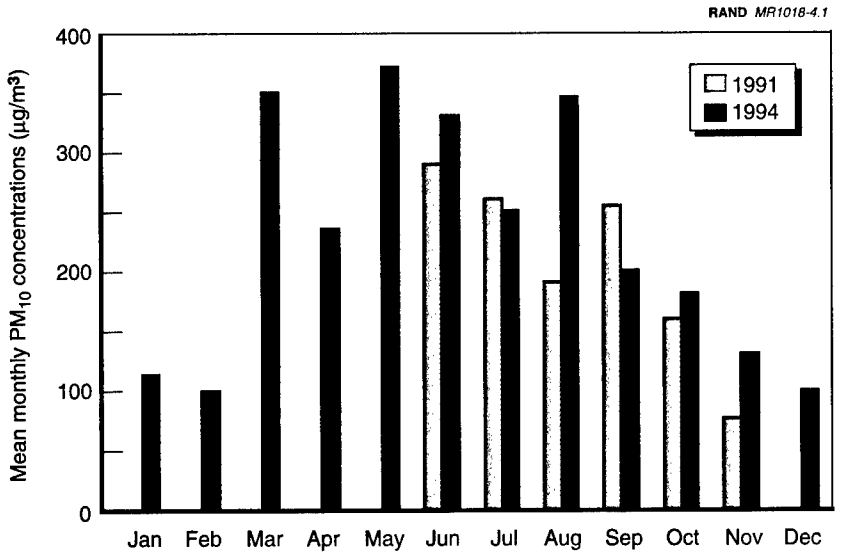


Figure 4.1—Mean Monthly Concentrations of PM_{10} in Camp Thunderock, Doha, Kuwait, in 1991 and in Kuwait City in 1994

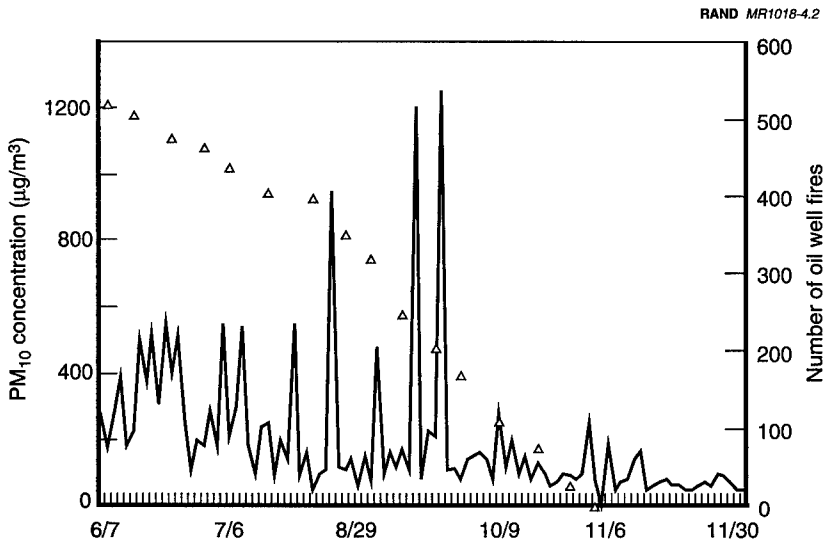


Figure 4.2—Daily PM_{10} Concentration in Camp Thunderock, Doha, Kuwait, from June to December 1991

either filtered in the nose or land in the mouth and throat and do not reach the lung. Particles smaller than 2.5 μm penetrate deeply into the lungs and can remain there for a long time.

The size distribution of the particles in the above-discussed ambient air samples were also measured. Figure 2.5 depicts the particle size distribution in air samples from Kuwait and Saudi Arabia. The analysis from these few samples indicates that there was a significant mass of particles in the size range that may have an effect on the respiratory system, particularly to those in sensitive or at-risk subgroups of the population. It is interesting to note that, within the military personnel stationed in Kuwait in 1994, during Operation Vigilant Warrior, there were more medical entries for respiratory codes than for orthopedic codes (USACHPPM, 1994).

Mostly because of its location, the Gulf War and its concomitant exposures could explain some of the respiratory complaints and diseases reported by its veterans. In particular, the exposures may explain the difference in asthma and bronchitis levels found in the Iowa Study between military personnel who went to the Gulf and those who did not. Figure 4.3 shows the time distribution of U.S. troops from August 1990 to August 1991, when most troops were redeployed from the Gulf, together with the monthly mean PM_{10} measured in 1991 and 1994. This graph shows that, when the PM_{10} concentrations started to

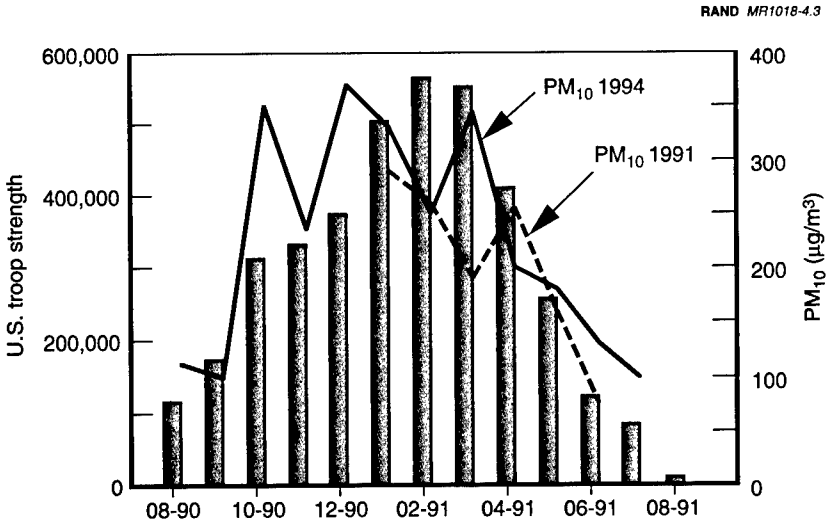


Figure 4.3—U.S. Troop Strength and PM_{10} Concentrations Versus Time

rise in March 1991, about 500,000 U.S. troops were in the region and could have been exposed to PM_{10} levels higher than the NAAQS for several months.

These results, together with the data in Figure 4.1, suggest that most of the exposures due to inhaled contaminants that had a potential for health risk were less likely to have come from the burning oil wells than to have come from the amount of very fine sand in the air.

A significant number of Gulf War veterans were smokers. Smokers, even young smokers, may react differently than nonsmokers to inhaled pollutants regardless of exposure levels. Also, a percentage of the veterans may have had some underlying predisposition to pulmonary disease that might have been triggered by exposure to high levels of PM_{10}. These high levels of PM_{10} may explain the preliminary findings of the Iowa Study regarding respiratory symptoms as well those reported in the CCEP.

Further research is needed on the health effects of exposure to multiple pollutants. Many questions need to be answered; for example, do the effects of inhaled particulate matter and other toxicants exceed those attributable to any one agent acting individually?

REFERENCES

ACGIH, TLVs and BEIs, 1997.

Ackerman-Liebrich, U., P. Leuenberger, J. Schwartz, et al., "Lung Function and Long Term Exposure to Air Pollutants in Switzerland. Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) Team," *Am J Respir Crit Care Med*, 155, 1997, pp. 122–129.

Agency for Toxic Substances and Disease Registry. Minimal Risk Levels (MRLs) for Hazardous Substances. 1997. http://ATSDR1.ATSDR.cdc.gov:8080/mrls.html.

Aksoy, M. and S. Erdem, "Follow-up Study on the Mortality and the Development of Leukemia in 44 Pancytopenic Patients with Chronic Benzene Exposure," *Blood*, 52, 1978, pp. 285–292.

Allred, E. N., E. R. Blecker, B R. Chaitman, et al., "Effects of Carbon Monoxide on Myocardial Ischemia," *Environ Health Perspect*, 91, 1991, pp. 89–132.

Andersen, I., G. R. Lundqvist, L. Molhave, O. F. Pedersen, D. F. Proctor, M. Vaeth, and D. P. Wyon, "Human Response to Controlled Levels of Toluene in Six-Hour Exposures," *Scan J Work Environ Health*, 9, 1983, pp. 405–418.

Aris, R., D. Christian, D. Sheppard, and J. R. Balmes, "The Effects of Sequential Exposure to Acidic Fog and Ozone on Pulmonary Function in Exercising Subjects," *Am Rev Respir Dis*, 143, 1991, pp. 85–91.

Bakan, A. S., A. Chlond, U. Cubasch, et al., "Climate Response to Smoke from the Burning Oil Wells in Kuwait," *Nature*, 351, 1991, pp. 367–370.

Bencko, V., "Chromium: A Review of Environmental and Occupational Toxicology," *J Hyg Epidemiol Microbiol Immunol*, 29, 1985, pp. 37–46.

Benignus, V. A., "Health Effects of Toluene: A Review," *Neurotoxicology*, 2, 1981, pp. 567–588.

Bennett, G. F., "Air Quality Aspects of Hazardous Waste Landfills," *Haz Wat Haz Mat*, 4, 1987, pp. 119–138.

Bhambhani, Y., R. Burnham, G. Snydmiller, I. MacLean, and T. Martin, "Effects of 5 PPM Hydrogen Sulfide Inhalation on Biochemical Properties of Skeletal Muscle in Exercising Men and Women," *Am Ind Hyg Assoc J*, 57, 1996, pp. 464–468.

Bhambhani, Y., R. Burnham, G. Snydmiller, and I. MacLean, "Effects of 10 PPM Hydrogen Sulfide Inhalation in Exercising Men and Women: Cardiovascular, Metabolic, and Biochemical Responses," *J Occup Environ Med*, 39, 1997, pp. 122–129.

Boey, K. W., S. C. Foo, and J. Jeyaratnam, "Effects of Occupational Exposure to Toluene: A Neuropsychological Study on Workers in Singapore," *Ann Acad Med Singapore*, 26, 1997, pp. 184–187.

Boyd, J. T., R. Doll, J. S. Faulds, and J. Leiper, "Cancer of the Lung in Iron Ore (Haematite) Miners," *Br J Ind Med*, 27, 1970, pp. 97–105.

Brain, J. D., J. Godleski, and W. Kreyling, "In Vivo Evaluation of Chemical Biopersistence of Nonfibrous Inorganic Particles," *Environ Health Perspect*, 102 (suppl 5), 1991, pp. 119–125.

Brand, P., J. Gebhart, M. Below, B. Georgi, and J. Heyder, "Characterization of Environmental Aerosols on Heligoland Island," *Atmos Environ*, 25 (PartA), 1991, pp. 581–585.

Braun-Fahrländer, C., U. Ackermann-Liebrich, J. Schwartz, H. P. Gnehm, M. Rutishauser, and H. U. Wanner, "Air Pollution and Respiratory Symptoms in Preschool Children," *Am Rev Respir Dis*, 145, 1992, pp. 42–47.

Brunnemann, K. D., M. R. Kagan, J. E. Cox, et al., "Determination of Benzene, Toluene and 1,3-Butadiene in Cigarette Smoke by GC-MSD," *Exp Pathol*, 37, 1989, pp. 108–113.

Burnett, R. T., R. E. Dales, M. E. Raizenne, D. Krewski, P. W. Summers, G. R. Roberts, M. Raad-Young, T. Dann, and J. Brook, "Effects of Low Ambient Levels of Ozone and Sulfates on the Frequency of Respiratory Admissions to Ontario Hospitals," *Environ Res*, 65, 1994, pp. 172–194.

Cahalan, R., "The Kuwait Oil Fires as Seen by Landsat," *J Geophys Res*, 97, 1992, pp. 565–571.

Chan, C. C., J. D. Spengler, H. Ozkaynay, and M. Lefkopoulou, "Commuter Exposures to VOCs in Boston, Massachusetts," *J Air Waste Manage Assoc*, 41, 1991, pp. 1594–1600.

Chen, L. C., P. D. Miller, M. O. Amdur, and T. Gordon, "Airway Hyperresponsiveness in Guinea Pigs Exposed to Acid-Coated Ultrafine Particles," *J Toxicol Environ Health*, 35, 1992, pp. 165–174.

Chen, Z., S. J. Liu, S. X. Cai, Y. M. Yao, H. Yin, H. Ukai, Y. Uchida, H. Nakatsuka, T. Watanabe, and M. Ikeda, "Exposure of Workers to a Mixture of Toluene and Xylenes. II. Effects," *Occup Environ Med*, 51, 1994, pp. 47–49.

Clarkson, T. W., "The Toxicology of Mercury," *Crit Rev Clin Lab Sci*, 34, 1997, pp. 369–403.

Coburn R. F., R. E. Foster, and P. B. Kane, "Considerations of the Physiological Variables That Determine the Blood Carboxyhemoglobin Concentrations in Man," *J Clin Invest*, 44, 1965, pp. 1899–1910.

Coombe, M. D., and S. F. Drysdale, "Assessment of the Effects of Atmospheric Oil Pollution in Post War Kuwait," *J R Army Med Corps*, 139, 1993, pp. 95–97.

Cowan, D. N., R. F. DeFraites, G. C. Gray, M. B. Goldenbaum, and S. M. Wishik, "The Risk of Birth Defects Among Children of Persian Gulf War Veterans," *New Engl J Med*, 336, 1997, pp. 1650–1656.

Danish, S., and I. M. Madany, "Concentrations of Nitrogen Dioxide Throughout the State of Bahrain," *Environ Pollut*, 77, 1992, pp. 71–78.

De Flora, S., A. Cam Oirano, M. Bagnasco, C. Bennicelli, G. E. Corbett, and B. D. Kerger, "Estimates of the Chromium(VI) Reducing Capacity in Human Body Compartments as a Mechanism for Attenuating its Potential Toxicity and Carcinogenicity," *Carcinogenesis*, 18, 1997, pp. 531–537.

Department of Defense, "Report to Congress on the Health Consequences of the Exposure of Persian Gulf Force Members to the Fumes of Burning Oil," 1993.

Devlin, R. B., W. F. McDonnell, H. S. Koren, and S. Becker, "Prolonged Exposure of Humans to 0.10 and 0.08 ppm Ozone Results in Inflammation in the Lung," 83rd Annual Meeting of the Air & Waste Management Association; June 1990; paper No. 90-150.2, 1990.

Devlin, R. B., W. F. McDonnell, R. Mann, S. Becker, D. E. House, D. Schreinemachers, and H. S. Koren, "Exposure of Humans to Ambient Levels of Ozone for 6.6 Hours Causes Cellular and Biochemical Changes in the Lung," *Am J Respir Cell Mol Biol*, 4, 1991, pp. 72–81.

Devlin, R. B., W. F. McDonnell, S. Becker, M. C. Madden, M. P. McGee, R. Perez, G. Hatch, D. E. House, and H. S. Koren, "Time-Dependent Changes of Inflammatory Mediators in the Lungs of Humans Exposed to 0.4 PPM Ozone for 2 Hr: A Comparison of Mediators Found in Bronchoalveolar Lavage Fluid 1 and 18 Hr After Exposure," *Toxicol Appl Pharmacol*, 138, 1996, pp. 176–185.

Dreschsler-Parks, D. M., J. F. Bedi, and S. M. Horvath, "Pulmonary Function Responses of Young and Older Adults to Mixtures of O_3, NO_2, and PAN," *Toxicol Ind Health*, 5, 1989, pp. 505–517.

Dusseldorp, A., H. Kruize, B. Brunekreef, P. Hofschreuder, G. deMeer, and A. B. van Oudvorst, "Associations of PM_{10} and Airborne Iron With Respiratory Health of Adults Living Near a Steel Factory," *Am J Respir Crit Care Med*, 152, 1994, pp. 1932–1939.

Echeverria, D. L., G. Fine, A. Langolf, A. Schork, and C. Sampio, "Acute Neurobehavioral Effects of Toluene," *Br J Ind Med.*, 46, 1989, pp. 483–495.

Elinder, C. G., T. Kjellstrom, C. Hogstedt, K. Andersson, and G. Spang, "Cancer Mortality of Cadmium Workers," *Br J Ind Med*, 42, 1985, pp. 651–655.

Environmental Protection Agency, *Evaluation of the Potential Carcinogenicity of Benzene*, review draft, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Carcinogen Assessment Group, OHEA-C-073-29, 1986.

Environmental Protection Agency, *Ambient Air Benzene Concentrations in 39 U.S. Cities, 1984–1986*, U.S. Environmental Protection Agency, Atmospheric Sciences Research Lab, EPA/600/D-87/160, Research Triangle Park, NC, 1987.

Environmental Protection Agency, *Guidelines for Exposure Assessment*, February 7, 1992.

Environmental Protection Agency, *Health Effects Assessment for Toluene.* Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, 1984.

Environmental Protection Agency, *National Air Quality and Emissions Trends Report.* US EPA, Office of Air Quality Planning and Standards. Research Triangle Park, NC, 1988.

Environmental Protection Agency, *National Ambient Air Quality Standards, 1997*, http://ttnwww.rtpnc.epa.gov/naaqsfin/naaqs.htm.

Etzell, R. A., and D. L. Ashley, "Volatile Organic Compounds in the Blood of Persons in Kuwait During the Oil Fires," *Arch Occup Environ Health*, 66. 1994, pp. 125–129.

Ferek, R. J., P. V. Hobbs, H. A. Herring, and K. K. Laursen, "Chemical Composition of Emissions from the Kuwait Oil Fires," *J Geophys Res*, 97, 1992, pp. 14,483–14,489.

Firket, J., "Fog Along the Meuse Valley," *Trans Faraday Soc*, 32, 1936, pp. 1192–1197.

Fishbeck, W. A., J. C. Townsend, and M. G. Swank, "Effect of Chronic Occupational Exposure to Measured Concentrations of Benzene," *J Occup Med*, 20, 1978, pp. 539–542.

Fishbein, L., "An Overview of Environmental and Toxicological Aspects of Aromatic Hydrocarbons III Xylene," *Sci Total Environ,* 43, 1985, pp. 165–183.

Fishbein, L., "Exposure from Occupational Versus Other Sources," *Scan J Work Environ Health,* 18(Suppl 1), 1992, pp. 5–16.

Gelzleichter, T. R., H. Witschi, and J. A. Last, "Synergistic Interaction of Nitrogen Dioxide and Ozone on Rat Lungs: Acute Responses," *Toxicol Appl Pharmacol,* 116, 1992, pp. 1–9.

Gerber, G. B., I. A. Leonard, and P. Jacket, "Toxicity, Mutagenicity and Teratogenicity of Lead," *Mutat Res,* 76, 1980, pp. 115–141.

Gordian, M. E., H. Ozkaynak, J. Xue, S. S. Morris, and J. D. Spengler, "Particulate Air Pollution and Respiratory Disease in Anchorage, Alaska," *Environ Health Perspect,* 104, 1996, pp. 209–297.

Gordon, T., L. C. Chen, J. M. Fine, R. B. Schlesinger, W. Y. Su, T. A. Kimmel, and M. O. Amdur, "Pulmonary Effects of Inhaled Zinc Oxide in Human Subjects, Guinea Pigs, Rats, and Rabbits," *Am Ind Hyg Assoc,* 54, 1992, pp. 503–509.

Graham, J. A., F. J. Miller, M. J. Daniels, E. A. Payne, and D. E. Gardner, "Influence of Cadmium, Nickel, and Chromium on Primary Immunity in Mice," *Environ Res,* 16, 1978, pp. 77–87.

Grandjean, P., "Human Exposure to Nickel," *IARC Sci Publ,* 53, 1984, pp. 469–485.

Greenberg, M. M., "The Central Nervous System and Exposure to Toluene: A Risk Characterization," *Environ Res,* 72, 1997, pp. 1–7.

Guidotti, T. L., "Occupational Exposure to Hydrogen Sulfide in the Sour Gas Industry: Some Unresolved Issues," *Int Arch Occup Environ Health,* 66, 1994, pp. 153–160.

Haldane, J., "Some Improved Methods of Gas Analysis," *J Physiol,* London, 22, 1898, pp. 465–480.

Hansen, J. C., and G. Danscher, "Organic Mercury: An Environmental Threat to the Health of Dietary-Exposed Societies?," *Rev Environ Health,* 12, 1997, pp. 107–116.

Hazucha, M. J., L. J. Folinsbee, E. Seal, and P. A. Bromberg, "Lung Function Response of Healthy Women After Sequential Exposures to NO_2 and O_3," *Am J Respir Crit Care Med,* 150, 1994, pp. 642–647.

Hemeon, W. C. L., The Estimation of Health Hazards from Air Pollution," *Arch Environ Health,* 11, 1955, pp. 397–402.

Hobbs, P. V., and L. F. Radke, "Airborne Studies of the Smoke from the Kuwait Oil Fires," *Science,* 256, 1992, pp. 987–991.

Hoek, G., and B. Brunekreef, "Acute Effects of a Winter Air Pollution Episode on Pulmonary Function and Respiratory Symptoms of Children," *Arch Environ Health*, 48, 1993, pp. 328–335.

Holmqvist, I. "Occupational Arsenical Dermatitis: A Study Among Employees at a Copper Ore Smelting Work Including Investigations of Skin Reactions to Contact With Arsenic Compounds," *Acta Derm Venereol*, 31 (suppl 26), 1951, pp. 1–214.

Horgan, J., "Burning Questions—Scientists Launch Studies of Kuwait's Oil Fires," *Sci Am*, 265, 1991b, pp. 17–24.

Horgan, J., "The Danger From Kuwait's Air Pollution," *Sci Am*, 265, 1991a, p. 30.

Hunt, W. "The Impact of the Kuwait Oil Fires—An Overview," Air and Waste Management Association Meeting, Kansas City, MO, June 1992, Paper 92-76.06.

Husain, T., "Kuwait Oil Fires: Regional Environmental Perspectives," *Pergamon*, 1995.

Inoue, Y., E. Barker, E. Daniloff, N. Kohno, K. Hiwada, and L. S. Newman, "Pulmonary Epithelial Cell Injury and Alveolar-Capillary Permeability in Berylliosis," *Am J Respir Crit Care Med*, 156, 1997, pp. 109–115.

International Agency for Research on Cancer, "IARC monographs on the Evaluation of Carcinogenic Risks to Humans: Vol. 58, Beryllium, Cadmium, Mercury, and Exposures in the Glass Manufacturing Industry," Lyon, France: World Health Organization, 1993.

Iowa Persian Gulf Study Group, "Self-Reported Illness and Health Status Among Gulf War Veterans. A Population-Based Study," *JAMA*, 277, 1997, pp. 238–245.

Iowa Persian Gulf Study Group, private communication, 1997.

Isa, M., H. Ali,. G. Ali, and A. Wright, "Satellite Monitoring of Smoke from the Kuwait Oil Fires," *J Geophys Res*, 97, 1992, pp. 551–563.

Ito, K., G. D. Thurston, C. Hayes, and M. Lippmann, "Associations of London, England, Daily Mortality with Particulate Matter, Sulfur Dioxide, and Acidic Aerosol Pollution," *Arch Environ Health*, 48, 1993, pp. 213–220.

Karl, H., and M. Leinemann, "Determination of Polycyclic Aromatic Hydrocarbons in Smoked Fishery Products from Different Smoking Kilns," *Z Lebensm Unters Forcsch*, 202, 1996, pp. 458–464.

Kelsey, K. T., F. Xia, W. J. Bodell, et al., "Genotoxicity to Human Cells Induced by Air Particulates Isolated During the Kuwait Oil Fires," *Environ Res*, 64, 1994, pp. 18–25.

Kerger, B. D., B. L. Finley, G. E. Corbett, D. G. Dodge, and D. J. Paustenbach, "Ingestion of Chromium(VI) in Drinking Water by Human Volunteers: Absorption, Distribution, and Excretion of Single and Repeated Doses," *J Toxicol Environ Health*, 50, 1997, pp. 67–95.

Khattak, M. N., "Natural and Anthropogenic Atmospheric Particulates at UPM Campus Dhahran, Saudi Arabia," *J Air Pollut Control Assoc*, 32, 1982, pp. 1153–1155.

Kilburn, K. H., and R. H. Warshaw, "Hydrogen Sulfide and Reduced-Sulfur Gases Adversely Affect Neurophysiological Functions," *Toxicol Ind Health*, 11, 1995, pp. 185–197.

Kipen, H. M., R. P. Cody, and B. D. Goldstein, "Use of Longitudinal Analysis of Peripheral Blood Counts to Validate Historical Reconstructions of Benzene Exposure," *Environ Health Perspect*, 82, 1989, pp. 199–206.

Kirkpatrick, J., personal communication, 1997.

Kjaergaard, S. K., and T. R. Rasmussen, "Clinical Studies of Effects of Nitrogen Oxides in Healthy and Asthmatic Subjects," *Cent Eur J Public Health*, 4(Suppl), 1996, pp. 23–26.

Kleinman, M. T., D. M. Davidson, R. B. Vandagriff, V. J. Caiozzo, and J. L. Whittenberger, "Effects of Short-Term Exposure to Carbon Monoxide in Subjects with Coronary Artery Disease," *Arch Environ Health.*, 44, 1989, pp. 361–369.

Knecht, E. A., W. J. Moorman, J. C. Clark, D. W. Lynch, and T. R. Lewis, "Pulmonary Effects of Acute Vanadium Pentoxide Inhalation in Monkeys," *Am Rev Respir Dis*, 132, 1985, pp. 1181–1185.

Koenig, J. Q., D. S. Covert, Q. S. Hanley, G. Van Belle, and W. E. Pierson, "Prior Exposure to Ozone Potentiates Subsequent Response to Sulfur Dioxide in Adolescent Asthmatic Subjects," *Am Rev Respir Dis*, 141, 1990, pp. 377–380.

Koenig, J. Q., D. S. Covert, S. G. Marshall, G. Van Belle, and W. E. Pierson, "The Effects of Ozone and Nitrogen Dioxide on Pulmonary Function in Healthy and in Asthmatic Adolescents," *Am Rev Respir Dis*, 136, 1987, pp. 1152–1157.

Koenig, J. Q., D. S. Covert, W. E. Pierson, Q. S. Hanley, V. Rebolledo, K. Dumler, and S. E. McKinney, "Oxidant and Acid Aerosol Exposure in Healthy Subjects and Subjects with Asthma, Part I: Effects of Oxidants, Combined with Sulfuric or Nitric Acid, on the Pulmonary Function of Adolescents with Asthma," *Res Rep Health Eff Inst*, 70, 1994, pp. 1–36.

Koenig, J. Q., D. S. Covert, M. S. Smith, G. Van Belle, and W. E. Pierson, "The Pulmonary Effects of Ozone and Nitrogen Dioxide Alone and Combined in Healthy and Asthmatic Adolescent Subjects," *Toxicol Ind Health*, 1988, pp. 521–532.

Koren, H. S., R. B. Devlin, S. Becker, R. Perez, and W. F. McDonnell, "Time-Dependent Changes of Markers Associated with Inflammation in the Lungs of Humans Exposed to Ambient Levels of Ozone," *Toxicol Pathol*, 19, 1991, pp. 406–411.

Koren, H. S., G. E. Hatch, and D. E. Graham, "Nasal Lavage as a Tool in Assessing Acute Inflammation in Response to Inhaled Pollutants," *Toxicology*, 60, 1990, pp. 15–25.

Kulle, T. J., J. H. Milman, L. R. Sauder, H. D. Kerr, B. P. Farrell, and W. R Miller, "Pulmonary Function Adaptation to Ozone in Subjects with Chronic Hitis," *Environ Res*, 34, 1984, pp. 55–63.

Lagerkvist, B., H. Linderholm, and G. F. Nordberg, "Vasospastic Tendency and Raynaud's Phenomenon in Smelter Workers Exposed to Arsenic," *Environ Res*, 39, 1986, pp. 465–474.

Lange, A., R. Smolik, W. Zatonski, et al., "Serum Immunoglobulin Levels in Workers Exposed to Benzene, Toluene, and Xylene," *Int Arch Arbeitsmed*, 31, 1973b, pp. 37–44.

Lawryk, N. J., P. J. Lioy, and C. P. Weisel, "Exposure to Volatile Organic Compounds in the Passenger Compartment of Automobiles During Periods of Normal and Malfunctioning Operation," *J Expo Anal Environ Epidemiol*, 5, 1995, pp. 511–531.

Lee, K. P., P. J. Gillies, "Pulmonary Response and Intrapulmonary Lipids in Rats Exposed to Bismuth Orthovanadate Dust by Inhalation," *Environ Res*, 40, 1986, pp. 115–135.

Leikauf, G. D., D. M. Spektor, R. E. Albert, and M. Lippman, "Dose-Dependent Effects of Submicrometer Acid Aerosol on Particle Clearance from Ciliated Human Lung Airways," *Am Ind Hyg Assoc J*, 45, 1984, pp. 285–292.

Levy, B. S., L. Hoffman, and S. Gottsegen, "Boilmakers' Bronchitis: Respiratory Tract Irritation Associated with Vanadium Pentoxide Exposure During Oil-to-Coal Conversion of a Power Plant," *J Occup Med*, 24, 1984, pp. 567–570.

Lewis, C. E., "The Biological Effects of Vanadium: II. The Signs and Symptoms of Occupational Vanadium Exposure," *AMA Arch Ind Health*, 19, 1959, pp. 497–503.

Lewtas, J., "Airborne Carcinogens," *Pharmacol Toxicol*, 72S, 1993, pp. 55–63.

Limaye, S., S. A. Ackerman, P. M. Fry, M. Isa, H. Ali, G. Ali, and A. Wright, "Satellite Monitoring of Smoke from the Kuwait Oil Fires," *J Geophys Res*, 97, 1992, pp. 14,551–14,554.

Linn, W. S., D. A. Shamoo, K. R. Anderson, R. C. Peng, E. L. Avol, and J. D. Hackney, "Effects of Prolonged, Repeated Exposure to Ozone, Sulfuric Acid,

and Their Combination in Healthy and Asthmatic Volunteers," *Am J Respir Crit Care Med*, 150, 1994, pp. 431–440.

Lodovici, M., P. Dolara, C. Casalini, S. Ciappellano, and G. Testolin, "Polycyclic Aromatic Hydrocarbon Contamination in the Italian Diet," *Food Addit Contam*, 12, 1995, pp. 703–713.

Madany, I. M., and E. Raveendran, "Polycyclic Aromatic Hydrocarbons, Nickel and Vanadium in Air Particulate Matter in Bahrain During the Burning of the Oil Fields in Kuwait," *Sci Total Environ*, 116, 1992, pp. 281–289.

Maenhaut, W., W. H. Zoller, and R. A. Duce, "Concentration and Size Distribution of Particulate Trace Elements in the South Polar Atmosphere," *J Geophys Res*, 84, 1979, pp. 2421–2431.

Major, J., G. Kemeny, and A. Tompa, "Genotoxic Effects of Occupational Exposure on the Peripheral Blood Lymphocites of Pesticide Preparing Workers in Hungary," *Acta Med Hung*, 49 (1–2), 1992, pp. 79–90.

Mason, H. J., A. G. Davison, A. L. Wright, et al., "Relations Between Liver Cadmium, Cumulative Exposure, and Renal Function in Cadmium Alloy Workers," *Br J Ind Med*, 45, 1988, pp. 793–802.

Mastrangelo, G., E. Fadda, and V. Marzia, "Polycyclic Aromatic Hydrocarbons and Cancer in Man," *Environ Health Perspect*, 104, 1996, pp. 1166–1170.

McMichael, A. J., "Carcinogenicity of Benzene, Toluene and Xylene: Epidemiological and Experimental Evidence," *IARC Sci Publ*, 85, 1988, pp. 3–18.

Minoia, C., S. Magnaghi, G. Micoli, M. L. Fiorentino, R. Turci, S. Angeleri, and A. Berri, "Determination of Environmental Reference Concentration of Six PAHs in Urban Areas (Pavia, Italy)," *Sci Total Environ*, 198, 1997, pp. 33–41.

Moeller, R. B., Jr., V. F. Kalasinsky, M. Razzaque, J. A. Centeno, et al., "Assessment of the Histopathological Lesions and Chemical Analysis of Feral Cats to the Smoke from the Kuwait Oil Fires," *J Environ Pathol, Toxicol and Oncol*, 13, 1994, pp. 137–149.

Mohsenin, V., "Effect of Vitamin C on NO_2-Induced Airway Hyperresponsiveness in Normal Subjects," *Am Rev Respir Dis*, 136, 1987, pp. 1408–1411.

Molfino, N. A., S. C. Wright, I. Katz, S. Tarlo, F. Silverman, P. A. McClean, J. P. Szalai, M. Raizenne, A. S. Slutsky, and N. Zamel, "Effect of Low Concentrations of Ozone on Inhaled Allergen Responses in Asthmatic Subjects," *Lancet*, 338, 1991, pp. 199–203.

Monarca, S., G. S. Sforzolini, and F. Fagioli, "Presence of Benzo[A]Pyrene and Other Polycyclic Aromatic Hydrocarbons in Suntan Oils," *Food Chem Toxicol*, 20, 1982, pp. 183–187.

Nadel, J. A., H. Salem, B. Tamplin, Y. Tokiwa, "Mechanism of Bronchoconstriction During Inhalation of Sulfur Dioxide," *J Appl Physiol*, 20, 1965, pp. 164–167.

Nasralla, M. M., "Air Pollution in the Semitropical Saudi Urban Area," *Environ Internat*, 9, 1983, pp. 255–264.

Needleman, H. L., A. Schell, D. Bellinger, A. Leviton, and E. N. Allred, "The Long-Term Effects of Exposure to Lead in Childhood: An 11-Year Follow-Up Report," *N Engl J Med*, 322, 1990, pp. 83–88.

Nettesheim, P., D. A. Creasia, and T. J. Mitchell, "Carcinogenic and Cocarcinogenic Effects of Inhaled Synthetic Smog and Ferric Oxide Particles," *J Natl Cancer Inst*, 55, 1975, pp. 159–169.

Nielsen, T., H. E. Jorgensen, J. C. Larsen, and M. Poulsen, "City Air Pollution of Polycyclic Aromatic Hydrocarbons and Other Mutagens: Occurrence, Sources and Health Effects," *Sci Total Environ*, 190, 1996, pp. 41–49.

NIOSH, USDHHS, CDC NIOSH, *Pocket Guide to Chemical Hazards*, U.S. Government Printing Office, 1997.

Norseth, T., "Cancer Hazards Caused by Nickel and Chromium Exposure," *J Toxicol Environ Health*, 6, 1980, pp. 1219–1227.

Orris, P., J. Cone, and S. McQuilkin, *Health Hazard Evaluation Report*, HETA 80-096-1359, Eureka Company, Bloomington, Illinois, Report No. 80-096; Cincinnati, OH: Department of Health and Human Services, National Institute for Occupational Safety and Health, 1983.

Pellizzari, E. D., "Analysis for Organic Vapor Emissions Near Industrial and Chemical Waste Disposal Sites," *Environ Sci Technol*, 16, 1982, pp. 781–785.

Peters, A., H. E. Wichmann, T. Tuch, J. Heinrich, and J. Heyder, "Respiratory Effects Are Associated with the Number of Ultrafine Particles," *Am J Respir Crit Care Med*, 155(4), 1997, pp. 1376–1383.

Petrucelli, B., personal communication, 1997.

Pinto, S. S., and C. M. McGill, "Arsenic Trioxide Exposure in Industry," presented at the 38th Annual Meeting of the Industrial Medical Association, April, 1953, Los Angeles, CA: *Ind Med Surg*, 22, 1953, pp. 281–287.

Pope, C. A. III, and D. W. Dockery, "Acute Health Effects of PM_{10} Pollution on Symptomatic and Asymptotic Children," *Am Rev Respir Dis*, 145, 1992, pp. 1123–1128.

Pope, C. A. III, and L. S. Kalkstein, "Synoptic Weather Modeling and Estimates of the Exposure-Response Relationship Between Daily Mortality and Particulate Air Pollution," *Environ Health Perspect*, 104, 1996, pp. 414–420.

Pope, C. A. III, J. Schwartz, and M. R. Ransom, "Daily Mortality and PM_{10} Pollution in Utah Valley," *Arch Environ Health*, 47, 1992, pp. 211–217.

Pope, C. A. III, "Respiratory Hospital Admissions Associated with PM_{10} Pollution in Utah, Salt Lake, and Cache Valleys," *Arch Environ Health*, 46, 1991, pp. 90–97.

Pope, C. A., III, "Particulate Pollution and Health: A Review of the Utah Valley Experience," *J Expo Anal Environ Epidemiol*, 6, 1996, pp. 23–34.

Raizenne, M., L. M. Neas, A. I. Damokosh, D. W. Dockery, J. D. Spengler, P. Koutrakis, J. H. Ware, and F. E. Speizer, "Health Effects of Acid Aerosols on North American Children: Pulmonary Function," *Environ Health Perspect*, 104, 1996, pp. 506–514.

Reddy, T. V., J. A. Stober, G. R. Olson, and F. B. Daniel, "Induction of Nuclear Anomalies in the Gastrointestinal Tract by Polycyclic Aromatic Hydrocarbons," *Cancer Lett*, 56, 1991, pp. 215–224.

Riley, J. J., N. G. Hicks, and T. L. Thompson, "Effect of Kuwait Oil Field Fires on Human Comfort and Environment in Jubail, Saudi Arabia," *Int J Biometeorol*, 36, 1992, pp. 36–38.

Roberts, J. M., R. S. Hutte, F. C. Fehsenfeld, et al., "Measurements of Anthropogenic Hydrocarbon Concentration Ratios in the Rural Troposphere: Discrimination Between Background and Urban Sources," *Atmos Environ*, 19, 1985, pp. 1945–1950.

Rosenthal, G. J., and C. A. Snyder, "Modulation of the Immune Response to Listeria Monocytogenes by Benzene Inhalation," *Toxicol Appl Pharmacol*, 80, 1985, pp. 502–510.

Rowe, D. R., K. H. Al-Dhowalia, M. E. Mansour, "Indoor-Outdoor Nitric Oxide and Nitrogen Dioxide Concentrations at Three Sites in Riyadh, Saudi Arabia," *J Air Waste Manage Assoc*, 41, 1991, pp. 973–976.

Rozen, M. G., and C. A. Snyder, "Protracted Exposure of C57BBL/6 Mice to 300 ppm Benzene Depresses B- and T-lymphocite Numbers and Mitogen Responses: Evidence for Thymic and Bone Marrow Proliferation in Response to Exposures," *Toxicology*, 37, 1985, pp. 13–26.

Rozen, M. G., C. A. Snyder, and R. E. Albert, "Depression in B- and T-lymphocyte Mitogen-induced Blastogenesis in Mice Exposed to Low Concentrations of Benzene," *Toxicol Lett*, 20, 1984, pp. 343–349.

Sasiadek, M., J. Jagielski, and R. Smolik, "Localization of Breakpoints in the Karyotype of Workers Professionally Exposed to Benzene," *Mutat Res*, 224, 1989, pp. 235–240.

Savolainen, H., "Biochemical and Clinical Aspects of Nickel Toxicity," *Rev Environ Health*, 11, 1996, pp. 167–173.

Schlesinger, R. B., J. E. Gorczynski, J. Dennison, L. Richards, P. L. Kinney, and M. C. Bosland, "Long-Term Intermittent Exposure to Sulfuric Acid Aerosol, Ozone and Their Combination: Alterations in Tracheobronchial Mucociliary Clearance and Epithelial Secretory Cells," *Exp Lung Res*, 18, 1992a, pp. 505–534.

Schlesinger, R. B., J. T. Zelikoff, L. C. Chen, and P. L. Kinney, "Assessment of Toxicologic Interactions Resulting From Acute Inhalation Exposure to Sulfuric Acid and Ozone Mixtures," *Toxicol Appl Pharmacol*, 115, 1992b, pp. 183–190.

Schlesinger, R. B, "Intermittent Inhalation of Nitrogen Dioxide: Effects on Rabbit Alveolar Macrophages," *J Toxicol Environ Health*, 21, 1987, pp. 127–139.

Schrenk, H. H., H. Heimann, G. D. Clayton, W. M. Gafafer, and H. Wexler, "Air Pollution in Donora, PA. Epidemiology of the Unusual Smog Episode of October 1948: Preliminary Report, Washington, DC: Public Health Service," *Pub Health Serv Bull No. 306*, Washington, D.C. PH Service, 1949.

Schwartz, J., D. W. Dockery, and L. M. Neas, "Is Daily Mortality Associated Specifically with Fine Particles?" *J Air Waste Manage Assoc*, 50, 1996, pp. 927–939.

Schwartz, J., "Air Pollution and Hospital Admissions for Respiratory Disease," *Epidemiology*, 7, 1996, pp. 20–28.

Schwartz, J., "Air Pollution and Hospital Admissions for the Elderly in Birmingham, Alabama," *Am J Epidemiol*, 139, 1994a, pp. 589–598.

Schwartz, J., "Air Pollution and Hospital Admissions for the Elderly in Detroit, Michigan," *Am J Respir Crit Care Med*, 150, 1994b, pp. 648–655.

Schwartz, J., "Particulate Air Pollution and Daily Mortality in Detroit," *Environ Res*, 56, 1991, pp. 204–213.

Schwartz, J., "PM_{10}, Ozone, and Hospital Admissions for the Elderly in Minneapolis, MN," *Arch Environ Health*, 49, 1994c, pp. 366–374.

Schwartz, J., "Short-Term Fluctuations in Air Pollution and Hospital Admissions of the Elderly for Respiratory Disease," *Thorax*, 50, 1995, pp. 531–538.

Schwartz, J., and D. Otto, "Blood Lead, Hearing Thresholds, and Neurobehavioral Development in Children and Youth," *Arch Environ Health,* 42, 1987, pp. 153–159.

Sentz, F. C., Jr., and A. B. Rakow, "Exposure to Iron Oxide Fumes at Arcair and Powder-Burning Operations," *Am Ind Hyg Assn,* 30, 1969, pp. 143–146.

Shah, J. J., and H. B. Singh, "Distribution of Volatile Organic Chemicals in Outdoor and Indoor Air," *Environmental Science and Technology,* 22, 1988, pp. 1381–1388.

Sharma, R., A. K. Haque, S. Awasthi, S. V. Singh, J. T. Piper, and Y. C. Awasthi, "Differential Carcinogenicity of Benzo[A]Pyrene in Male and Female CD-1 Mouse Lung," J *Toxicol Environ Health,* 52, 1997, pp. 45–62.

Sherwin, R. P., and V. Richters, "Centriacinar Region (CAR) Disease in the Lungs of Young Adults: A Preliminary Report," 1991, in Bergland, R. L., D. R. Lawson, and D. J. McKee, eds. *Tropospheric Ozone and the Environment: Papers from an International Conference,* March 1990, Los Angeles, CA, Pittsburgh, PA, Air and Waste Management Association, pp. 178–196. (A&WMA Transaction Series, no TR-19.)

Singh, G. B., L. J. Salas, K. B. Cantrell, et al., "Distribution of Aromatic Hydrocarbons in Ambient Air," *Atmos Environ,* 19, 1985, pp. 1911–1920.

Sjoberg, S. G., "Vanadium Pentoxide Dust: A Clinical and Experimental Investigation on Its Effect After Inhalation," *Acta Med Scand,* 238S, 1950, pp. 1–18.

Sorahan, T., "Mortality from Lung Cancer Among a Cohort of Nickel Cadmium Battery Workers: 1946–84," *Br J Ind Med,* 44, 1987, pp. 803–809.

Spektor, D. M., V. A. Hofmeister, P. Artaxo, J. A. P. Brague, F. Echelar, D. P. Nogueira, C. Hayes, G. D. Thurston, and M. Lippmann, "Effects of Heavy Industrial Pollution on Respiratory Function in the Children of Cubatao, Brazil: A Preliminary Report," *Environ Health Perspect,* 94, 1991, pp. 51–54.

Spektor, D. M., G. D. Leikauf, R. E. Albert, and M. Lippmann, "Effects of Submicrometer Acid Aerosols on Mucociliary Transport and Respiratory Mechanics in Asymptomatic Asthmatics," *Environ Res,* 37, 1985, pp. 174–191.

Stevens, R., J. Pinto, Y. Mamane, et al., "Chemical and Physical Properties of Emissions from Kuwait Oil Fires," *Wat Sci Tech,* 27, 1993, pp. 223–233.

Sunderman, F. W., "A Review of the Metabolism and Toxicology of Nickel," *Ann Clin Lab Sci,* 1977, pp. 377–398.

Teculescu, D., and A. Albu, "Pulmonary Function in Workers Inhaling Iron Oxide Dust," *Int Arch Arbeitsmed,* 31, 1973, pp. 163–170.

Thun, M. J., T. M. Schnorr, A. B. Smith, W. E. Halperin, and R. A. Lemen, "Mortality Among a Cohort of U.S. Cadmium Production Workers—An Update," *J Natl Cancer Inst*, 74, 1985, pp. 325–333.

Thurston, G. D., K. Ito, C. G. Hayes, D. V. Bates, and M. Lippmann, "Respiratory Hospital Admissions and Summertime Haze Air Pollution in Toronto, Ontario: Consideration of the Role of Acid Aerosols," *Environ Res*, 65, 1994, pp. 271–290.

Toft, K., T. Olofsson, A. Tunek, et al., "Toxic Effects on Mouse Bone Marrow Caused by Inhalation of Benzene," *Arch Toxicol*, 51, 1982, pp. 295–302.

Townsend, J. C., M. C. Ott, and W. A. Fishbeck, "Health Exam Findings Among Individuals Occupationally Exposed to Benzene," *J Occup Med*, 20, 1978, pp. 543–548.

United Kingdom Ministry of Health, *Mortality and Morbidity During the London Fog of December 1952*, London, United Kingdom: Her Majesty's Stationary Office, 1954, (Reports on public health and medical subjects, No. 95).

U.S. Army Environmental Hygiene Agency, *Kuwait Oil Fire Health Risk Assessment, Interim Report*, Aberdeen Proving Ground, MD: U.S. Army Environmental Hygiene Agency, 1992.

U.S. Army Environmental Hygiene Agency, *Kuwait Oil Fire Health Risk Assessment: Final Report*, No. 39-26-L192-91, U.S. Army Environmental Hygiene Agency, 1994.

U.S. Army Center for Health Promotion and Preventive Medicine, "Vigilant Warrior 94," 1994.

Utell, M. J., M. W. Frampton, P. E. Morrow, C. Cox, P. C. Levy, D. M. Speers, and F. R. Gibb, "Oxidant and Acid Aerosol Exposure in Healthy Subjects and Subjects with Asthma. Part II: Effects of Sequential Sulfuric Acid and Ozone Exposures on the Pulmonary Function of Healthy Subjects and Subjects with Asthma," Cambridge, MA: Health Effects Institute, research report no. 70, 1994, pp. 37–93

Wallace, L. A., "Major Sources of Benzene Exposure," *Environ Health Perspect*, 82, 1989a, pp. 165–169.

Wallace, L. A., "The Exposure of the General Population to Benzene," *Cell Biol Toxicol*, 5, 1989b, pp. 297–314.

Wallace, L. A., E. D. Pellizzari, T. D. Hartwell, et al., "Personal Exposure, Indoor-Outdoor Relationships, and Breath Levels of Toxic Air Pollutants Measured for 355 Persons in New Jersey." *Atmos Environ*, 19, 1985, pp. 1651–1661.

Wallace, L., W. Nelson, R. Ziegenfus, E. Pellizzari, L. Michael, H. Zelon, T. Hatwell, R. Perrit, and D. Westerdahl, "The Los Angeles TEAM Study: Personal Exposures, Indoor-Outdoor Air Concentrations, and Breath Concentrations of 25 Volatile Organic Compounds," *J Expo Anal Environ Epidemiol*, 1, 1991, pp. 157–192.

Warren, D. L., D. J. Guth, and J. A. Last, "Synergistic Interaction of Ozone and Respirable Aerosols on Rat Lungs. Part II. Synergy Between Ammonium Sulfate Aerosol and Various Concentrations of Ozone," *Toxicol Appl Pharmacol*, 84, 1986, pp. 470–479.

Warren, J. B., and N. Dalton, "A Comparison of the Bronchodilator and Vasosupressor Effects of Exercise Levels of Adrenaline in Man," *Clin Sci*, 64, 1983, pp. 475–479.

Wester, R. C., H. I. Maiach, L. D. Gruenke, et al., "Benzene Levels in Ambient Air and Breath of Smokers and Nonsmokers in Urban and Pristine Environments," *J Toxicol Environ Health*, 18, 1986, pp. 567–573.

Wood, J. A., and J. L. Porter, "Hazardous Pollutants in Case II Landfills," *J Air Pollut Control Assoc*, 37, 1987, pp. 609–615.

Yardley-Jones, A., D. Anderson, D. P. Lovell, et al., "Analysis of Chromosomal Aberrations in Workers Exposed to Low-Level Benzene," *Br J Ind Med*, 47(1), 1990, pp. 48–51.

Zenz, C., and B. A. Berg, "Human Responses to Controlled Vanadium Pentoxide Exposure," *Arch Environ Health*, 14, 1967, pp. 709–712.